NATIONAL INSTITUTE OF SIDDHA

Tambaram Sanatorium, Chennai - 47

AFFILIATED TO THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI - 600 032

A study on

VENPADAI

(DISSERTATION SUBJECT)

For the partial fulfillment of the requirements to the Degree of

DOCTOR OF MEDICINE (SIDDHA)

BRANCH IV – KUZHANTHAI MARUTHUVAM DEPARTMENT

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Need For The Study

Even through vilitigo is a pathological disorder, the community causes a Major psychological stress to the victims.

There are some evidences from the individual trials, recorded short time benefit from steroids besides its side effects.

The methods currently available to treat vitiligo are largely unsatisfactory.

More cure the patients walk in to our national institute of siddha, with this vitiligo are children's, particularly female, it seems the patients are particularly from kalpakkam to kuduvancherry. Hence it is a need to do a study to make on impact on

1.INTRODUCTION

The word siddha comes from the word siddhi which means an perfection or heavenly bliss. The siddhars were further the greatest scientists in ancient times. they were men of highly cultured intellectual and spiritual faculties combined with super nature powers

Agathiya siddhar who is the chief of the siddhars school is said to have been a celebrated philosopher and physician who laboured amongst the tamils in southern india

The entire Siddha or Tamil medicine system consists of three great subdivisions namely

a. Noyilla Neri (Preventive)
b. Noy Neeku Neri (Curative methods)
c. Uramakkumurai (Strengthening Methods)

Prevention can mostly save our body and soul but modernization results in diseased condition. Siddha system is playing major role in treating chronic diseases.The Siddha system is the holistic system of codified life style a health care perfected many thousands of years ago in the Tamil speaking peninsular India.The granite rocks of the southern peninsula belong to the category of Archaic Rocks and are estimated to be around 2.5 to 4 billion years old.This system is also called as Tamil Maruthuvam, Chinthamani Maruthuvam and Arivan Maruthuvam. Since the advent of Thirumoolar who wrote the classic Thirumanthiram this is popularly called as Siddha system and practiced by Siddhars – the enlightened seekers.

Siddha system clearly lays down the general principles of body constituents in the classic Bootha system. They hold that the universe is a macrocosm made up of the five primordial elements or Boothas, viz Nilam (Earth), Neer (Water), Thee (Fire), Vali (wind) and Veli (Space) and the human being is a microcosm made up of the same five elements.
The Siddha system is divisible in to three major divisions, which are totally interwoven.

They are

1. Kappu (Prevention and Protection)
2. Neekam (Remove of diseases and disability)
3. Nirappu (Restoration of people to their full potential of good health)

A specific feature in Siddha care is the Kalpamurai also called as kayakalpa where the human being from the stage of foetus to around 80 years of age can be fortified in to rock like strength through serious of efforts with lifestyle, strengthening drugs and formulations.
Siddha system characterizes the basic guna of human beings

Sathuva gunam – Saint like attitude

Rasathuva gunam – King like attitude

Thamova gunam – Lesser endowmends - lower level of attainment both materially and mentally

Any person may have varying propositions these guna's in himself or herself.
Noi illa neri is the special approach of the siddha system where regular dietary habit, early rising, physical and mental disciplinaries are all emphasized.

Man is nothing but a world in miniature containing the fine elements and all the various principles which constitute the mineral the vegetable and the animal kingdoms venpudai is one of the most important skin disorder encountered in medical practice in india it depresses the patient mentally to a great deal, venpudai is also called 'swetha kuttam' which is one of the 18 varieties of skin disorders, under kuttam noted in yugi chithamani.

There are many misgivings about this diseases in the minds of the public.

- It is still a wrong notion of the people that it is contagious and it will lead to leprosy because of the name venkuttam and also believe that it is due to sin and karma.

- So the author decided to use form 'venpudai' instead of venkuttam for this clinical activity.

- The disease beard social stigma school children having venpudai are struggling to study.

- In the treatment of skin disease as "the siddha system of medicine has wonderful drugs' the author choice of drugs for the clinical study is
- Lagu seena chooranam- internal with hotwater.
- Venpadai pattru with –external cows urine

2.AIM AND OBJECTIVES

AIM :

Diseases of the skin are a common occurrence. They account for a great deal of misery, suffering, incapacity and economic loss. Venpadai affects the patients physically and mentally.

The clinical study of Venpadai was done in selected cases of both the gender and they were treated in the Inpatient ward in the Ayothidoss Pandithar Hospital of National Instutite of Siddha, Tambaram Sanatorium, Chennai - 47.

OBJECTIVES :

- To have an idea of the incidence of this disease with gender, age, occupation, social status, diet and seasonal variations.
- Collection and detailed study of various literatures, dealing with definition, aetiology, classification, signs and symptoms, prognosis, treatment and diet for venpadai.
- To correlate the signs and symptoms of venpadai with that of modern science.
- To do bio-chemical analysis and toxicological study of the trial drug.
- To find out the efficacy of lagu seena chooranam and venpadai pattru(venkutta lapanam) (external) in venpadai patients.
- To find out the side effects or adverse reactions if any.

PROTOCOL

A PILOT OPEN CLINICAL TRIAL OF *LAGU SEENA CHOORANAM* **AND** *VENPADAI PATTRU(VENKUTTA LABANAM)* **FOR THE TREATMENT OF** *VENPADAI (VITILIGO)*

By

Dr.A.Satheesh kumar.M.D(S), P.G.Student, DEPT.OF KUZHANTHAI MARUTHUVAM, NIS,CHENNAI.

1. INTRODUCTION

Vitiligo is an acquired idiopathic depigmentary condition, which though world wide in distribution, is most common in India. It is a source of great social embarrassment to dark - skinned people. It affects all age groups with no predilection to either sex. Its incidence is markedly increased.

In Siddha system of medicine, the drugs which are commonly used in the treatment of *Venpadai* includes *karpogi choornam* and *karpogi thylam.*

In *Agathiar pillai tamil* text, and *Siddha vaithiya patharthaguna vilakkam(kannusamiyam)* text, there is a preparation for Venpadai, whose efficacy is not well known. So, I try to evaluate it in a pilot clinical trial in our OPD and IPD patients in National Institute of Siddha, Chennai.

2. AIM

To find out the efficacy of *Lagu seena Chooranam* (Internal) and *venpadaipattru(Venkutta labanam)* (External) in Venpadai patients.

1.To find out the side effects of the drugs, if any.

2.Biochemical Study

3.Toxicological Study.

3. POPULATION AND SAMPLE

The population consists of all patients with Venpadai (completely depigmented or hypo-pigmented patches without any structural change in the skin) satisfying the inclusion and exclusion criteria mentioned below.Sample consists of Venpadai patients attending the OPD of Ayothidoss Pandithar Hospital of the National Institute of Siddha, Chennai – 47.

4. SAMPLE SIZE

The trial size will be 50 patients.

5. INCLUSION CRITERIA

1. Age between 5 to 12 years.
2. Willing to give specimen of blood for the investigation when required.
3. Willing to be admitted in the Hospital for 48 days or willing to attend OPD once in 7 days for 48 days.

6. EXCLUSION CRITERIA

Patients of Jaundice, hypo–pigmented patches of leprosy and burns, connective tissue disorders, and heart ailments are not eligible for this trial.

7. WITHDRAWAL CRITERIA

1. Any drastic changes occurring in hematological/immunological findings during treatment period.
2. Development of any exacerbation in clinical features.
3. Occurrence of any serious illness

8. TRIAL DRUG, DOSAGE, DURATION

INTERNAL DRUG: *Lagu seena Chooranam1-2 gm* with hotwater twice a day after food.

EXTERNAL DRUG :*venpadai pattru(Venkutta labanam)*-10gm with cows urine applied twice a day

TRIAL PERIOD : 48 days

9. ASSESSMENT AND TESTS

Clinical assessment

Site ,Size,Colour,Margin,Shape,Itching.

Investigations

Blood Test: RBC, Hb, Tc, Dc, ESR

SIDDHA ASPECTS

1. **ENVAGAI THERVU**

   Naa, Niram,Mozhi,Vizhi,Sparism,Naadi,Malam,Moothiram

2. **NEERKURI**

   Niram,Manam,Nurai,Enjal,Eadai.

3. **NEIKURI**

10. CONDUCT

Venpadai patients satisfying the inclusion and exclusion criteria will be included in the trial. Informed consent will be obtained from the patients. A day before starting the trial treatment, cleaning of mukkutras by purgation will be carried out by *Siddhathi ennai,* early morning 1 ml with hot water.

For O.P patients, the trail drugs will be issued for 7 days at a time. They will be asked to attend the clinic once in 7 days. Also, they will be instructed to bring back unconsumed trail drugs and return them during their next clinic visit.

Tests will be carried out before treatment and at the end of the treatment. Photos will be taken before and after treatment.

11. FORM

Form I – Selection proforma

It is used before admission of the patients to the trial

Form II – Assessment form

It is used once in 7 days during treatment.

12. ANALYSIS

Cure will be mainly assessed by comparing the before and after treatment photos. Quantitative parameters will be assessed by paired t-test.

4. REVIEW OF LITERATURE

SIDDHA ASPECTS

SYM: *Venpadai* (In *yugi'*s classification, it is under 18 types of *kuttam* as *swethakuttam*)
Swethakuttam (Swetha – White)

WHEN TO SUSPECT/RECOGNSISE (DIGNOSTIC CRITERIA):

- *Venpadai* is a chronic skin disease characterized by various sizes of hypopigmentation of the skin.
- In siddha literatures if the lesions may be present in palms, palms and region (soles, anal region), genitalia are uncurable
- The colour of the patches look like burns is also uncurable.

"தடிப்பாகத் தவளநிறம் போல் வெளுத்துச்
சர்வாங்கமும் வெளுத்தாற்றான் றிரும்பும்
மடிப்பாக மயிர்வெளுத்தா லசாத்ய மாகும்
வரிவுதடுவுள் ளங்கைக்குதங்குய்யந்
நெடிப்பாக நெருப்புப்பட்டது போற்புண்ணாய்
நிறமிருந்தா லசாத்தியமென்றே யுரைக்கலாகும
வெடிப்பாக மேனியெல்லாம் வெளுத்து வீங்கில்
வெண்சுவேத குட்டமென்றே விளம்ப லாமே."

யூகி சிந்தாமணி - 800

நோய் வரும் வழி (Aetiology)

According to Siddha System, the predisposing causes for this disease have been described as hereditary factor, stress and strain, malnutrition and venereal exposure and no other specific causes have been mentioned for venpadai.

According to Thirumoolar Karukkidai Vaithiya Nool

“வியாதியுண் மூவாறு விளங்கிய குட்டங்கேள்

சுயாதிக் கிரந்தி சுழன் மேகத்தாலாறும்

பயாதி மண்ணுளப் பலவண்டினா லெட்டும்

நியாதி புழுநாலாய் நின்றதிக் குட்டமே”

Six types of Kuttam i.e. skin diseases are caused by kirandhi and megam, eight types are caused by insects in the soil, and four types are caused by worms.

Excessive heat and cold, laziness, excessive sleep in day time, unbridled sexual indulgence, robbery etc. These habits are supposed to be the factors which lower the immunity of the body (Iyarkkai vanmai) and make it vulnerable to the disease.

Excessive intake of food items which are hard to digest, imbalanced food, vomiting and frequent intake of food mixed with stone and hair.

Prolonged mental depression, intention to spoil others, raping, greed, use of indescent and disrespectful words against God and highly religious and noble people, neglecting or

phans and beggars, cursing the elders and so on have also been given as predisposing causes by Yughi Text.

According to Agasthiyar Kanma Kandam

“சேர்ந்த குட்டமொடு குறைநோய்கள் வந்த

சேதி கேள் மலராத வரும்பு கொய்தல்

தாரிந் சீவசெந்து வதைகள் செய்தல்

தாய்தந்தை மனதுநொந்து ரோந் தானே

தானென்ற தெய்வரு தனையழித்தல்

சார்வான பெரியோர்கள் தமைப் பழித்தல்

கானென்ற நந்தவனம் பூஞ்செடிகள் வெட்டல்

கருமமடா சரீ ரத்திற் காசுபோலே

ஊனென்ற வுடம்பெல்லாம் பொட்டு பொட்டா

யுடன் வெளுத்து குறைநோயுதிரஞ் சிந்தும்

வானென்ற கருமங்கள் தீர்ப்பதற்கு

வகையொன்று சொல்வேன் கேள்."

Kuttam may be hereditary.

Apart from all other etiological factors kuttam is also considered as 'Karuma noi'.

Classification

According to Yugi Chinthamani

"முத்தாகும் குட்டந்தான் பதினெட்டுக்கும்

முனியான யூகிநான் சொல்லக் கேளாய்

புத்தாகும் புண்டரீக குஷ்டத்தோடு

பொருகின்ற விற்போடகக் குட்டமாகும்

பத்தாகும் பாமா குஷ்ட ஏகசர்ம குஷ்டம்

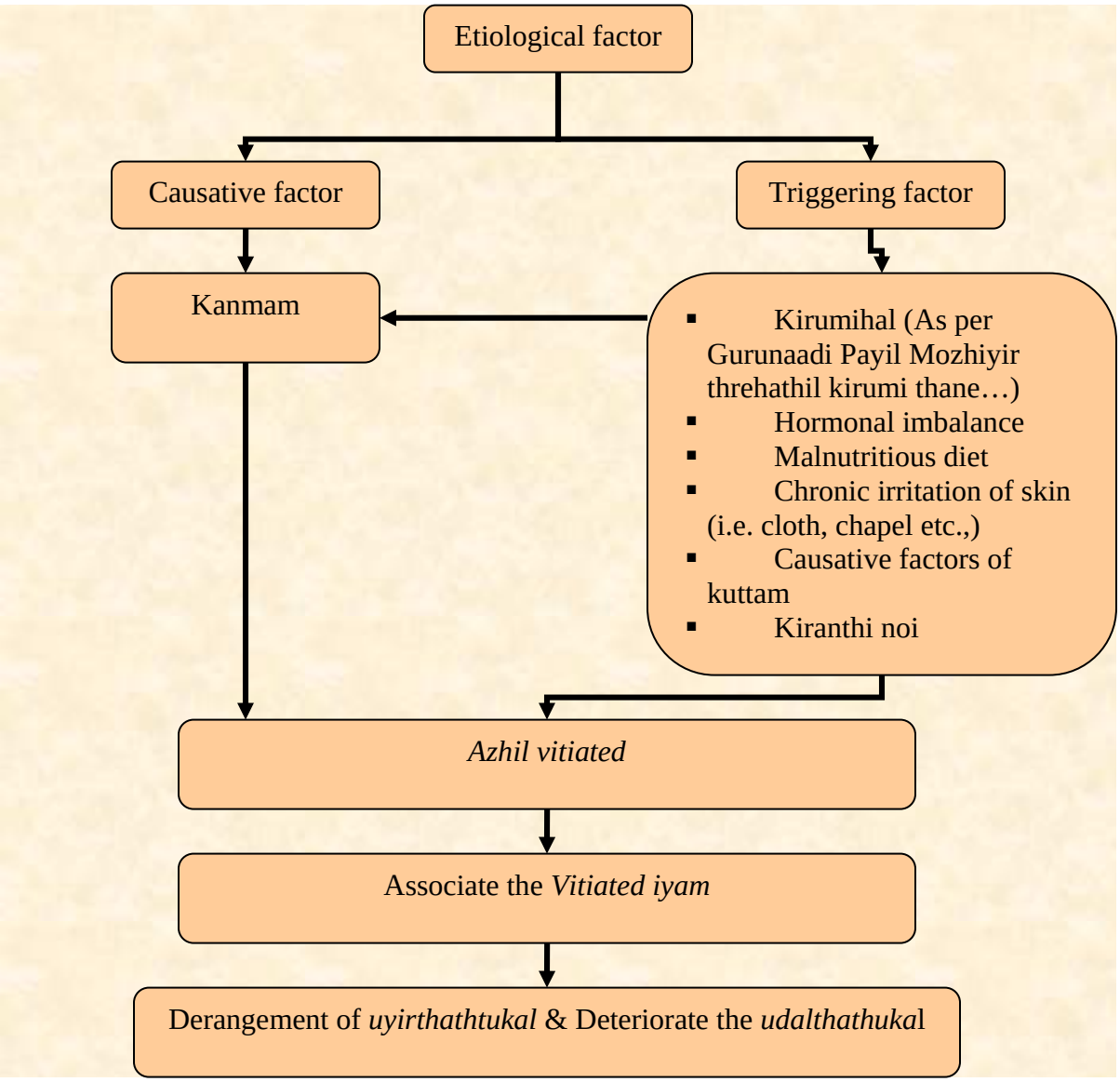
பரிவான கர்னகுஷ்டம் சர்மகுஷ்டங்

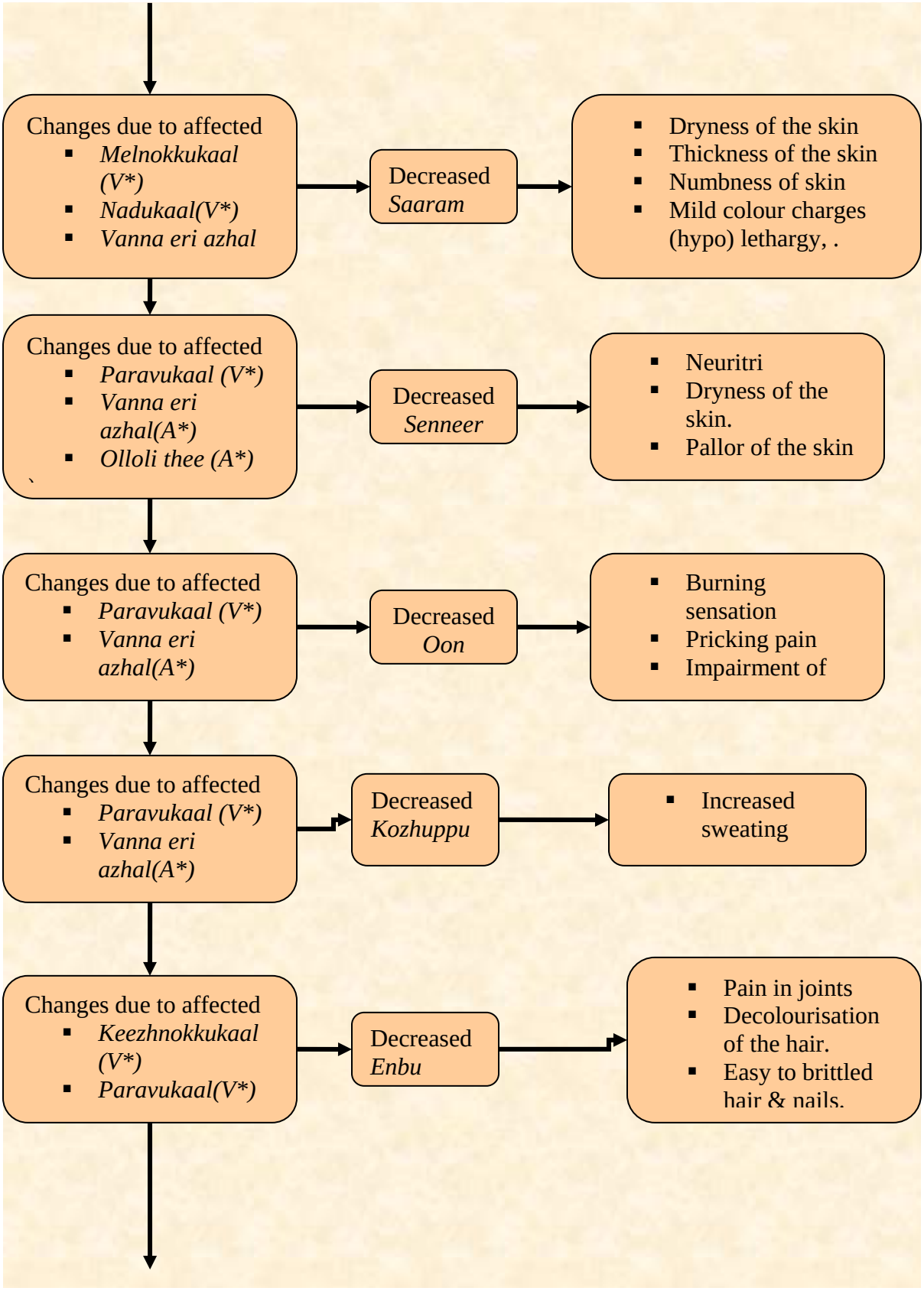
கித்தாகுங் கிருஷ்ண குட்டம் அவுதும்பர குட்டம்

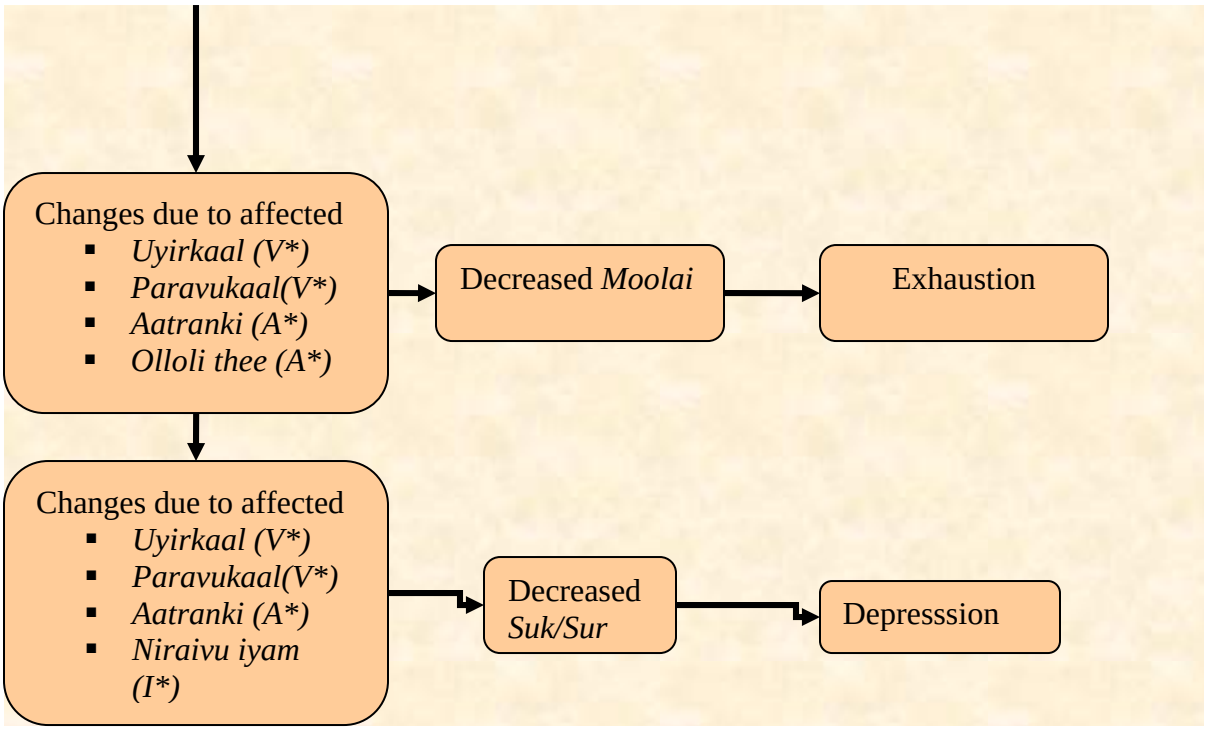
கேடியான மண்டல குஷ்டமாகு மென்னே

குட்டமா மபரிச குஷ்ட மோடு

SIGNS AND SYMPTOMS BASED ON *UYIR AND UDAL THATHUKKAL*




Changes due to affected
Melnokkukaal (V*)
Nadukaal(V*)
Vanna eri azhal
Decreased Saaram
Dryness of the skin
Thickness of the skin
Numbness of skin
Mild colour charges (hypo) lethargy, .
Changes due to affected
Paravukaal (V*)
Vanna eri azhal(A*)
Olloli thee (A*)
Decreased Senneer
Neuritri
Dryness of the skin.
Pallor of the skin
Changes due to affected
Paravukaal (V*)
Vanna eri azhal(A*)
Decreased Oon
Burning sensation
Pricking pain
Impairment of
Changes due to affected
Paravukaal (V*)
Vanna eri azhal(A*)
Decreased Kozhuppu
Increased sweating
Changes due to affected
Keezhnokkukaal (V*)
Paravukaal(V*)
Decreased Enbu
Pain in joints
Decolourisation of the hair.
Easy to brittled hair & nails.



மருவலாங் கிடீப குஷ்டந் சர்மதல குஷ்டந்

திட்டமாற் தத்துரு குஷ்ட மோடு

தக்கான சித்துமா குஷ்டஞ் சதாரு குஷ்டந்

துட்டமாஞ் சுவேத குஷ்டதன் னோடொக்கச்

சுயம்பான பதினெட்டுக் குட்டமாச்சே.”

1. Pundareeka kuttam
2. Virpodaka kuttam
3. Baama kuttam
4. Gaja saruma kuttam
5. Karna kuttam
6. Siguram
7. Krishna kuttam

8. Avudhumbaram

9. Mandala kuttam

10. Abarisa kuttam

11. Visarchika kuttam

12. Vibaathika kuttam

13. Kideeba kuttam

14. sarmathala kuttam

15. Thethru kuttam

16. Sithuma kuttam

17. Sathaaru kuttam

18. Swetha kuttam

According to Siddhar Aruvai Maruthuvam

Venpadai has been classified into 3 types on the basis of Mukkutram, they are,

1. Vaatha venpadi
2. Pittha venpadai
3. Kaba venpadai

According to Siddha Maruthuvam Sirappu :

Venpadai has been classified into 4 types :

1. Vaatha venpadai
2. Pittha venpadai
3. Kaba Venpadai
4. 4. Mega venpadai

According to Athma Rakshamirtha Vaidhya Sarasankiraham

Venpadai is classfied int 4 types :

1. Venkuttam
2. Senkuttam
3. Karunkuttam

4. Peru viyathi

Clinical Features of Eighteen Kutta Rogam

According to Thanvanthiri Vaithyam

“மீக்கௌத் தோறுமெலுமோர் முகம் வெளுக்குமாகில்

நோக்கியல் மரிக்குஞ் சொன்ன வெண்குட்டமாமே”

Discolouration of skin into white

A. According to Vaithya Saarasangiraham - sole, hands, lips, scalp, fingers and wrist joint - all these organs are found with white coloured patches which are circumscribed along with thickened border and gradually spread which is known as “Venpadai”. Blood, muscle and adipose tissue are affected by disease.

Discolouration of hairs, absence of normal skin texture comparing the adjoining normal skin area and appearance of burns is not curable.

B. According to Pararaasa Sekaram

1. Watery discharge
2. Gray Colour
3. Foul Smelling

According to Anubhava Vaithiya Deva Ragasiyam

“இந்நோயை குஷ்டமென கூறினும் இது குஷ்ட வகைகளினின்று வேறுபட்டது என்பதையும் குஷ்டத்தைப் போல் அவ்வளவு கொடுமையான வியாதி அல்லவென்றும் உணர வேண்டும்.

இந்நோயில் திட்டு திட்டாக வெண்மை நிறமான படைகள் உண்டாகி பிறகு தேகம் முழுவதும் பரவி உடலை விகாரப்படுத்துதல் முதலிய குணங்களை உடையது.

Three Types

1. Vaatha Venpadai
2. Piththa Venpadai
3. Kaba Venpadai - Indian Medicine P. 143

Clinical Features :

1. The Skin appears glittering and rough
2. There is an excessive perspiration or no perpiration
3. Discolouration
4. Heat and itching of the skin
5. Numbness in some parts of the body.

According to Sirappu Maruthuvam

1. Vaatha Venpadai
2. Pitha Venpadai
3. Kaba Venpadai
4. Mega Venpadai

1. Vaatha Venpadai

It is characterised by the depigmented patches, which are dry, rough, reddish with some what pale-black in colour.

2. Pittha Venpadai

It is characterised by the depigmented patches red in colour like lotus flower, spreading with burning sensation and loss of hairs on that area.

3. Kaba Venpadai

It is characterised by the depigmented patches white in colour like leucus flower,spreads with rashes and itching

4. Mega Venpadai

It is due to the venereal diseased and it occurs after 4 or 6 months after the onset as disease, syphilis within four or six months of the attack. This venpadai develops initially along the nape and the adjoining spaces. Also gradually it affects the shoulder joints, back of trunk.Clinical features of this type are clearly defined by the author of “Siddha Maruthuvam Sirappu” as follows:

Depigmented patches are small in number, pale in colour, with turmeric colour or dark colour margin marked with hyperpigmented signs.These lesions are circumscribed with 2 mm to 3 mm diameter or above.This correct picture of hypopigmented and hyper-pigmented skin seems to be more or less a multi eyed filter (sieve - like).

Females are more prone to this Mega Venpadai, Therefore anti-syphilitic therapy is mandatory in the early period of the treatment.

Character of Venpadai

Skin colour will change to reddish black or reddish white or white colour with spreading nature.The imbalance of the three thathus produces certain lesions in skin known as kuttam.

Absence of perspiration and thickening of skin may produce the colour changes in skin.

தீரும், தீராதவை

In Thanvanthiri Vaithiyam

சாத்தியம் - 11

“பூண்டந் நுரவினோடு சதாரிகம் புண்டரீ கந்த

தாண்டு விற்போடம் பாமாவுடன்மைதலம் வெண்குட்டம்

கூண்டிடு காகறந்தி சிறுமை யசல குட்டம்

வேண்டியவியாதியோடும் பதினொன்றும் விரித்துக் காணே.”

அசாத்தியம் - 7

“சொல்லுகுட்டம் ஏழுவகைபேர் சொல்லிக் கபால சர்மீகம்

வெல்லு முதும்பா மேகிடிபம் விசர்ச்சிமண்டலக் கிரமும்

மல்லல் தருமீசி யகுவை யாகும் பெயரோ ரேழாகும்

வல்லகியாதிக் குணமதனை வகுத்துப் பாரிலுறுரைப்பேனே”.

Curable - 11

1. Thethuru kuttam
2. Sadhaaru kuttam
3. Pundareega kuttam
4. Virpodaga kuttam
5. Sarma thala kuttam
6. Baama kuttam
7. Kaha nandhi
8. Venkuttam
9. Sithuma kuttam
10. Alasakuttam
11. Vibaathiga kuttam

Incurable - 7

1. Kabaala kuttam
2. Sarumamega kuttam
3. Avudhumbara kuttam
4. Kideeba kuttam
5. Visarchika kuttam
6. Aguvai kuttam
7. Mandala kuttam

In Yugi Chinthamani - 800

“குட்டந்தான் பதினெட்டில் சாத்தியந்தான்

கூறக்கேள் விற்போடக பாமா குட்டம்

திட்டந்தான் கெசசர்ம குட்டமொடு

கிருட்டின குட்டமவுதும்பர குட்டந்தானும்

திட்டமாந் தேதிதிருக் குட்டமொடு

செய்சித்துமா குட்டங் கிடிப குட்டம்

தட்டந்தான் மிகுந்த சதாரு குட்டம்

சமகிருட்ண குட்டம் சாத்தியமா மென்னே”

Curable - 10

1. Virpodaka kuttam
2. Baama kuttam
3. Gaja saruma kuttam
4. Krishna kuttam

5. Avuthumbara kuttam

6. Thethuru kuttam

7. Sithuma kuttam

8. Kideepa kuttam

9. Sathaaru kuttam

10. Sarmathala kuttam

Incurable - 11

1. Pundareeka kuttam

2. Karna kuttam

3. Sikura kuttam

4. Mandala kuttam

5. Abarisa kuttam

6. Visarchika kuttam

7. Swetha kuttam

முக்குற்ற வேறுபாடுகள் (Pathology)

According to Noi Nadal Noi Muthal Nadal Thirattu and Siddha Maruthuvam texts in Siddha System the manifestation of all diseases are the result of derangement of dhoshas that is vaatha piththa and kaba.

According to the theory of Siddha the human body is composed of 96 thathuvams (of constituent principles in nature including panchaboothams and thridhosam). The Siddha System of Medicine is based onthe thridhosha theory. This includes the three humors vaatham, pitham and kabam. These three humours are primary and essential consitutional factors of human body. These factors exist in 1 : $^{1}/_{2}$: $^{1}/_{4}$ ratio

respectively in the normal body. This hormonial existence is responsible for the proper functioning of the body. Any alteration in the above ratio can cause disease in the body like, vaatha disease, pitha disease and kaba disease.

Combination of five elements in muthathu :

Vaatham	=	Vali	+	Vin
Piththam	=	Thee		
Kaba	=	Mann	+	Neer

MUTHATHU IYAL OR UYIR THATHU IYAL:

The most important clinical approach of the Siddha physicians is to assess the status and function of the three uyir thatus. This is described as Naadi sothanai and Naadi nadai Arithal.

a) **Vali – (Kattru + Veli)**
b) **Azhal – (Thee)**
c) **Iyyam – (Neer+Mann)**

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1:½:¼) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

The three thathus are manifest at the wrist and are individually and collectively assessed.

These three humour are divided in to various types and have their functions specifically.

VALI:

According to the physiological function vali is of ten types. They are

1. Uyir Kaal (pranan)

This is the first of ten vital airs. According to Yugi muni, pranan starts from moolatharam and comes through the nostrils and causes the act of inspiration and expiration. The inspiration and expiration are not equal and their ratio is 8:12. The process helps in the digestion of ingested food.

2. Kizhnokkumkaal(Abanan)

Apanan, the downward air, starts from swathittanam and descends towards the pelvis and is responsible for excretion of urine and faeces. This is green in color. It contracts the anus. It helps to take the essence of the digested food to the different parts of the body which requires food. The god attributed is varadarajan.

3**. Paravukaal(Vyanan)**

Vyanan arises from the shoulders and go through all the 72,000 nerves and thus activate voluntary and involuntary movements of the body and thus make them to extend the contract. This appreciates the sense of touch, helps to take the essence of the food to the strategic points of the body and guards the body.

4. Melnokkumkaal (Udhanan)

Udhanan starts from the umbilical region (udarakkini) and takes the essence of food and stations it at appropriate places. It helps in digestion and assimilation of food.

5. Nadukkal (Samanan)

Samanan starts from the umbilical cord and spread out up to the lowerlimb. This is responsible for the balance of the other four vathas. It equalizes the six tastes, water, food etc and helps in assimilation.

6. Naagan

Naagan is responsible for higher intellectual functions, hearing, thinking etc. It causes closing and opening of the eye lids.

7. Koorman

Koorman starts from the mind and causes winking of the eyelids, yawning and closure of mouth. It gives strength and helps to visualize things and causes lacrimal secretion

8. Kirukaran

Kirukaran lies in the tongue and causes nasal and salivary secretions. It induces hunger. Sneezing and cough are attributed to kirukaran. It is black in color

9. Devadaththan

Laziness is attributed to devadaththan. Occular movements and human passions are attributed to this vatham. It stays either at the anus or at urinary orifice.

10. Dhananjeyan

Dhananjeyan functions from the nose and it is responsible for the bloating of the body after death and also for the foul smell.

AZHAL:

It is not just heat in our body. Functionally this human bodies warmth/heat of life is divided in to five types. They are

1. Akkuanal (Analagam)

It lies between the stomach and the intestine and causes digestion and dries up moist ingested substances

2. Vanna eri (Ranjagam)

This fire lies in the stomach and gives red color to the chyme and produces blood. It improves blood.

3. Attralangi (Sadhagam)

This fire lies mainly in limbs. It gives energy for activities

4. Nokku Azhal(Alosagam)

It lies in the eyes and causes the faculty of vision. It helps to visualize things

5. Ollolithee (Prasagam)

It gives color and complexion and brightness to the skin

IYYAM:

It is of five types. They are

1. Alli Iyyam (Avalambagam)

It lies in the lungs and helps in respiration. It causes firmness of the limbs. This is vital among all types of kapham for it controls the other four kapham and maintains equilibrium

2. Neerpi Iyyam (Kilethagam)

It lies in the stomach. It mixes the consumed food and water and promotes the digestive process

3. Suvaikanna Iyyam (Pothagam)

It lies in the tongue and helps to realize the taste of the consuming food.

4. Niraivu Iyyam (Tharpagam)

Sustaining in the head, this gives refrigerant effect to cool the eyes and other sense organs.

5. Ondri Iyyam (Sadhigam)

Sustaining in the joints this makes them more freely and easily.

Since venpadai patients are not having defined description of pathology, the pattern of disturbance in Vali, Azhal, Iyyam keeps on varying. The manifestation of these uyir thathus keep on changing according to the predominant symptom. But alteration in azhal thathu is seen mostly.

EZHU UDAL KOORUGAL:

SEVEN PHYSICAL CONSTITUENTS

The human body is made of seven basic, physical constituents. These constituents should be in harmony and function normally. Any variation in them will lead to their functional deviations.

The Natural characters of the seven physical constituents

1. **Saaram** (Chyle) : This gives mental and physical perseverance
2. **Senner** (Blood) : Imparts color to the body, nourishes the body and is responsible for the ability and intellect of an individual
3. **Oon** (Muscle) : It gives shape to the body according to the physical activity and covers the bones.
4. **Kozhuppu** (Adipose tissue): It lubricates the joints and other parts of the body to function smoothly
5. **Enbu** (Bone) : Supports the frame and responsible for the postures and movements of the body
6. **Moolai** (Bone marrow): It occupies the medulla of the bones and gives strength and softness to them
7. **Sukkilam/Suronitham** (Sperm and Ovum) : It is responsible for reproduction

THE VARIATIONS OF THE PHYSICAL CONSTITUENTS

1. SAARAM

Increased Saaram : Leads to diseases of increased kapham like indigestion Etc

Decreased Saaram : Leads to loss of weight, tiredness, lassitude, dryness of the skin and diminished activity of the sense organs.

2. SENNEER

Increased Senneer : Causes boils in different parts of the body throbbing pain, anorexia, mental disorder, splenomegaly,Colicky pain., increased blood pressure, reddish eye and Skin, jaundice, haematuria etc.

Decreased Senneer : Leads to anaemia, tiredness, neuritis and lassitude, Pallor of body.

3.OON

Increased Oon : Oon in excess causes cervical lymph adenitis, venereal ulcer, tumour in face, abdomen, thigh genitalia etc are the signs of increased Oo Decreased Oon : Leads to impairment of sense organs, joints jaw, thigh and genitalia gets shortened

4. KOZHUPPU

Increased Kozhuppu: Identical to that of increased Oon associated with Dyspnoea and loss of acidity Decreased Kozhuppu: Leads to pain in the hip region and diseases of the spleen

5. ENBU

Excess Enbu : Growth in bones and teeth Decreased Enbu : Loosening of teeth and nails and Splitting and falling of hair

6. MOOLAI

Increased Moolai : Causes heaviness, swollen eyes, swollen phalanges, Oliguria and non healing ulcers Decreased Moolai : Causes osteoporosis and sunken eyes

7. SUKKILAM / SURONITHAM

Excess Sukkilam/Suronitham : Causes lust towards women and cause Urinary calculai

Decreased Sukkilam/Suronitham : Causes failure in reproduction, pain in the genitalia.

ENVAGAI THERVU

Nowadays advanced diagnostic tools have been developed by modern bio-medical scientists. But Siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

1.NAA

Signs and symptoms in the tongue are considered here. Size, appearance, thickness, color(pigmented, magenta) fissured (longitudinal, transverse) coated, geographical patches, oral hairy leucoplakia, candida, apthus ulcers, sense of taste, saliva secretion

2.NIRAM

The color of skin is mainly considered here but also the change in other organs.

3. MOZHI

The change in the normal sound of voice mainly uratha olli (Valithel), thazhntha olli (Melithal), physiological and mental status can also be noted during conversation.

4. VIZHI

Color, warm, burning sensation, irritation, visual perception

5. PARISAM

Observations by touch, temperature, sensory impairment, masses, nodes, swelling, texture of the skin, pain, hardness, odematus, and dullness shall be noted.

6.MALAM

The stools are examined for quantity, hardening(malakattu) loose motion (beethi) color, smell

7. MOOTHIRAM

Neer Kuri : Urine is to be obsorbed for the following characters

Niram(color)

Alavu(quantity)

Edai (specific gravity)

Manam (smell)

Nurai (froth)

Enjal (deposit)

NEI KURI

A unique traditional method for diagnosis with urine is Neikkuri. Urine is freshly collected in a clean glass vessel and a drop of Gingili oil is dropped in to it. The gingelley oil should be prepared by wooden press. Machine pressed oil is not an effective tool.

Mode of spreading is noted, usually

Vali noi - Aravu (Snake like spread)

Azhal noi - AAzhi (Ring like spread)

Iyyam -Muthu (Pearl like spread)

venpadai starts with disturbed Azhal and eventually involves all the three uyir thathus – thus resulting in various patterns of oil spread in the urine surface.An unique pattern, is seen mostly like star fish with branches and sieve (plate with pores), irregular border, speed of spread is also to be noted. It has been observed that the initial star shaped spread reduces as the patients responds to treatment.

MANAGEMENT OF VENPADAI

REJUVENATION:

KALPA MARUNTHU:

Pothukalpam(General):

- *Ponnangaani karpam* (Alternanthera sessilis) (Used as recipes along with *Milagu* (Piper nigvum), *Kariuppu* (Sodium chloride)).

Sirappu:

- Kittikizhangu (Acalypha fruticosa) used as daily dishes like *curry, vatral* etc.,.
- Lemon used as pickle or juices (to be continued for six months).
- *Ayapirungaraja karpam*
- *Ayajambeera karpam*

KALPA YOGAM:

- *Sarvangaasana* is most useful for this complained *Shirasana* is also useful, where as the other asanam have been included for general health and fitness.
- *Pranayamam*

The common benefits of yogam

Padmaasanam

Uses

The constant practice of this asana brings about good digestive power, cheerfulness and balancing of mukkutram.

Sarvangaasanam

Uses

It prevents narai, thirai and moopu. (i.e.) prevents ageing. By stimulating the thyroid gland it gives strength to all the organs of the body. It cures venpadai.

UNAVU (DIET):-

To be add:

- **Bitter tasted foods**

Greens:

- *Sirukeerai* (Amarantus tricolor) – “Kanpuhaichal.....Pongumpitham.....Sirukeerai thanai kol.”
- *Pannai keerai* (Celosia argentea) - Pannai yilankeerai yathu...Karappan sirangu pun maatrum.....
- Paruppu keerai (Postulaca oleracea) – “Pillai parupilaiku pithamarum.....”
- Keerai thandu (Amaranthus gangeticus) – Senkeerai thandathu than theeratha pithathai thengaamal oati vidum...
- Agathi keerai (Sesbania grandiflora) – Pithmathu saanthiya hum agathiyilai thinuubavaiku…

To be avoid:

- Sour, spicy, salty foods
- Curd, oils, alcohol, sugar, non – vegetarian diet
- Ulundhu (Vigna mango)
- Mustard
- Brinjal.

Other advices:

- Oleation: Oil bath should be taken twice a week is advisable.
- Be add bitter tasted herbs like Azadhirachta indica, Acacia cateche etc.,
- *Savuri pazha thylam*
- Palasu uppu (salt of Butea monosperma) – For external application
- De-oleaginous substances:- The powdered form of Iluppai pinnakku (Bassia longifolia).
- Eechaam paai (a type of mattress prepared from the leaves of Phoenix sylvestris)

Food stuffs that bring the vaatha, piththa and kaba dhoshas to the normal physiological level have to be consumed.

MODERN ASPECTS

The Skin

Dermatology is the study of skin diseases. Disease of the skin, are a common occurrence, account for a great deal of misery, suffering in capacity and economic loss. A very few skin diseases are contagious.

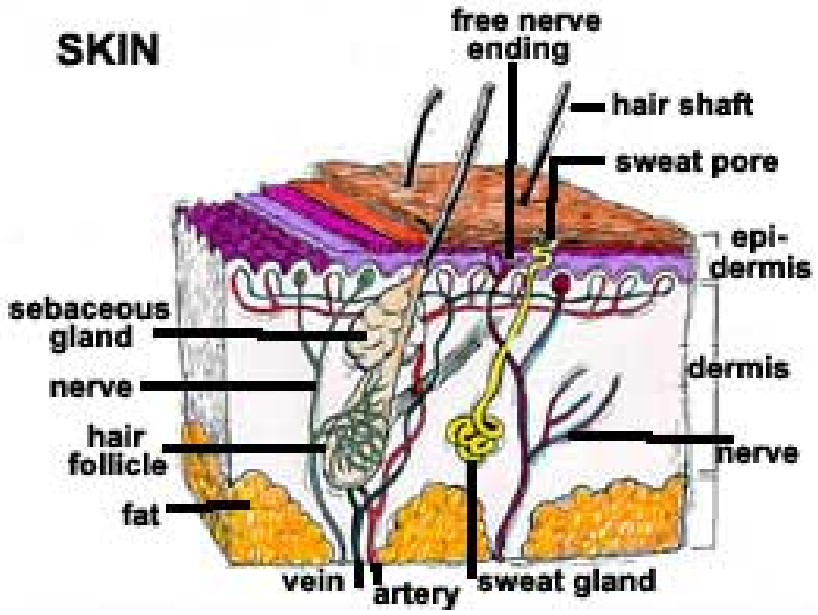
Hippocrates, Father of Medicine described many skin diseases and divided them into two groups according to their exogenous or endogenous causes. He attributed the origin of disease to abnormal mixing of black and yellow bile, blood and phlegm. The theory of adnormally mixed humors played a major role in dermatology for along time.

Dermatology is a branch of medicine dealing with the skin. Its roots reach back to antiquity. The obviously manifested skin diseases have drawn the attention of men since time immemorial.

Skin Anatomy

The human skin is the outer covering of the body and is continous with the mucous membrances in the region of the mouth, nose, urogential organs and the anus. In an adult the skin surface measures 1.5 - 2 m^2 while the thickeness of the skin varies from fractions of a millimeter to 4 mm. The thickeness of the epidermis varies from 0/06 - 0.9 mm to 0.5 - 0.6 mm. The thickness of the subcutaneous fat varies considerably. Some areas are devoid of fat while in others (on the abdomen and gluteal regions), it is several centimeters thick. The mass of skin of an adult accounts for approximately 5 % while together with subcutaneous fat for about 10 to 17.7 % of the total body mass.

Normal Skin Structure



The colour of the skin may change because the amount of the pigment in it varies due to external and internal factors.

The skin surface is covered with hairs over a great area. The areas devoid of hairs are the lips, palms and soles,glans penis, inner surface of the prepuce and the inner surface of the labia majorum and minorum.

Skin Histology

The skin develops from two germinative zones. The ectoderm which is represented by the epidermis (the most superficial skin layer) and the mesoderm (the midele embryonal layer) represented by two layers namely the true skin, or dermis (the middle layer) and the subcutaneous fat or hypoderm the deepest skin layer.

The boundary between the epidermis and dermis forms a wavy line because of the presence of skin papilla (special out growth on the surface of the true skin) the spaces between which are filled with epithelial processes.

Vascular System of Skin

Vascular system of the skin is formed of several networks of blood vessels. Large arterial vessels stretch from the bascia through the subcutaneous fat and give off small branches to the fat lobules. On the boundary of the dermis and hypoderm, they divide into branches which stretch horizontally and anastomose with one another. A deep arterial plexus of skinforms, which gives rise to branches supplying the holes of the sweat glands, the hair follcles and the fat lobules. The epidermis is devoid of blood vessels. The most powerful network of blood vessels is located in the skin of the face, palms, soles, lips, genitals and in the skin around the anus.

Lymphatic system of skin

The lymphatic system of the skin forms superficial and deep networks. The superficial lymphatic network arises on the papillary layer as blind rounded dilated capillaries between which there are numerous anastamoses. The second network of lymph vessels is in the lower part of the dermis and already has valves. There is a network of wide loops forming lymphatic plexus and deeper parts are continous with lymph trunks

Epidermis

The epidermis is stratified epithelium undergoing keratinization; it consists of the following layers :

i) Germinative layer of stratum basale

ii) Prickle - cell layer or stratum spinosum

iii) Granular layer or stratum granulosum

iv) Stratum lucidum

v) Horny layer or stratum corneum

There are many nerve endings in the epidermis but no blood vessels and the cells are supplied with nutrients by the lymph flowing in the intercellular slits.

Dermis

The dermis is located between the epidermis and the subcutaneous fat. Two layers are distinguished in it, the papillary or subepithelial layers and the reticular layer. The papillary layer is that part of the dermis which is found betwen the epidermis and the superficial network of blood vessels. The reticular layer merges with the subcutaneous fat and is not demarcated from it sharply.

The Deep Part of the Skin

The subcutaneous tissue or hypoderm consists of thick bundles of collagen and elastic fibres stretching from the regular dermal layer and forming a wide loop reticulum in which accumulations of large fat cells, lobules or fatty tissues are lodged. The fat cells are almost completely formed of a large drop of fat which displace the cell nucleus to the periphery and a very small amount of protoplasm.

Vitiligo

The name ‘vitiligo’ is derived from the Latin word skin eruption, victim meaning a blemish (spoil the beauty of) happens to be a synonym for it.

White skin is the literal meaning of leucoderma, derma being derived from the Greek words, leucas and dermis. Leucas means white and dermis means skin.

Celeus was the first Roman physician of the 2nd century to coin the word vitilligo, because the disease resembles the white patches of a spotted calf (vitelus).

Vitiligo is characterized by the presence of non-pigmented areas of irregular shape, which develop on the epidermis of skin and hair. In this condition there is absence of deficiency of melanin, a dark pigment of the skin produced by melancytes under the stimulation of the sun light and possibly, under the control of a melanin stimulating hormone of the hypophysis.

It is also regarded to develop through eczema scar of prick by injection needle, injury by burn or from other accidents, by friction of foot, wearing tight clothes. It has also been observed in persons who have suffered serious illness due to typhoid, jaundice, liver diseases, diabetes, worms, constipation and diarrhoea.

The non pigmented patches whitish or reddish are round or oval in shape with smooth surface and slowly grow into large, irregularly outlined areas. It may be the result of skin diseases or it may be a harmless condition of unknown cause.

Definition

Vitiligo is a disorder of the skin especially due to loss of pigment without any disturbances and textural alterations.

A condition due to failure of melanin formation in the skin produces sharply demarcated, milky white patches with hyperpigmented borders.

But Leucoderma is an acquired no inherited (incurred as a s result of factors acting from or originating outside the organism). Condition with localized loss of pigmentation of the skin. Inheritance means an acquisition of characters or qualities by transmission from parent or off spring.

It is an extremely common depigmentary disorder of great medicosocial significance among the dark people, aetiology is uncertain association with variable penetrance; no age is except, both sexes. A symptomatic puncture linear, oval, circular or irregular, discrete or confluent depigmented and or hypopigmented macules on otherwise normal skin is confined to mucocutaneous functions dermatomal unilateral or bilateral, symmetrical or asymmetrical generalized or universal over laying hair retain pigment or turn white, no autonomic or sensory disturbanes, sub burn or chronic solar damage in longstanding cases, impredictable and capricious course, stationary, self healing or progressive.

It is quite clear that vitiligo is due to some derangement in the pigment metabolism resulting in appearance of white patches in the skin. It is hard to say whether the site of derangement is usually general or local, but the main affected part is the skin, which is the most exposed part of the body. It can be examined by naked eye and can furnish a lot of information about the person and the diesease. In certain cases the changes are not clear. Hence the study of the skin structure and its physiology is essentialssss for proper asssessment.

Epidemiology

Vitiligo is an acquired idiopathic depigmentary condition which, though worldwide in distribution, is most common in India, Egypt and other tropical countries. It is a source of great social embarassment of dark-skinned people. It affects all age groups with no predilection to either sex.

Gross Anatomical Changes in Vitiligo

Vitiligo represents an acquired patchy loss of pigments of the skin. There are no gross changes seen except irregularly demarcated, depigmented patches of varying size, usually surrounded by hyperpigmented skin. These are seen distributed symmetrically or asymmetrically at various parts of the body.

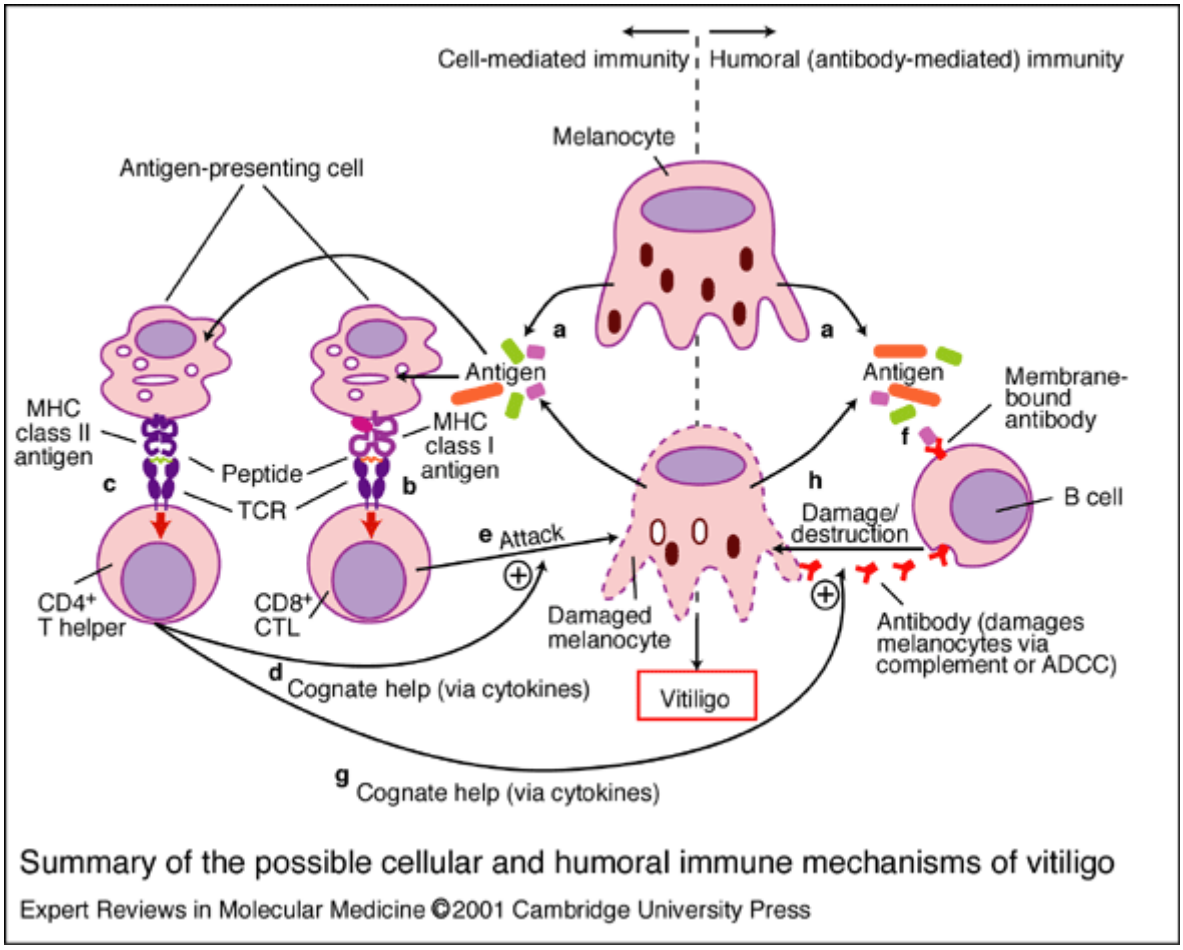
Histopathalogic changes in Vitiligo

Marked histological changes do not occur in cases of vitiligo. All the layers of the epidermis and dermis appear normal except a few changes which can be seen after special stains.

In the affected area the basal cells and the keratinzing cells of the other layers of epidermis do not contain melanin pigment granules in them. The contrast can be seen at a junction of the normal and vitiligenous areas of the skin, especially by silver staining or DOPA reaction. The pigment cells, the melanocytes are not seen in the affected area but they are present in the adjacent normal skin. At the border of the patches of vitiligo the melanocytes often appear large and posses long dendritic process filled with melanin granules. Electron microscopic studies confirm the absence of melanocytes in areas of long standing vitiligo.

There are collections of mononuclear cells at dermo epidermal junction at the border between vitisliginous and normal skin. These cells are predominately small lymphocytes. In the long standing cases where the skin has become thick and scaly, varying amount of the keratosis is seen.

Possible cellular and humoral immune mechanisms of vitiligo:



Summary of the possible cellular and humoral immune mechanisms of vitiligo

Expert Reviews in Molecular Medicine ©2001 Cambridge University Press

Melanin

Melanin - Derived from the Greek word Melas, meaning black.

Melanin is endogenous nonhaemoglobin derived brown or black pigment formed when the enzyme tyrosinase catalyses the oxidation of tyrosin to dihydroxy phenylalanine (DOPA) in melancocytes.

Distribution

It is widely distributed in the body but peculiarly enough it is limited only to those structures which have got an ectodermal origin, for eg: skin, hair, choroid coat of retina and substanita nigra of the brain. It is formed from tyrosine by oxidation,metabolism and polymerization.

Pigmentation of the Skin

The colour of the skin may be brown or even black according to the amount of pigment present.

Even in white races most parts of the skin contain brown pigment granules in the deepest layers of the germinative zone of the epidermis. In dark races they are more abundant and extend throughout the whole zone.

Functions

The function of melanin in the choroids is namely to convert the eye ball into a perfect dark chamber. Since nervous tissue is derived from ectoderm, the melanin in the substantia nigra may represent the vestigeal remnants of the melanin in the substantia nigra may represent the vestigal remnants of the melanin forming properties. Melanin is the great protector of the skin against the actinic rays of the sun.

Melanin Formation

Melanin, wherever it is found, is formed in the local cells by the enzyme tyrosinase (or) melanase. The mother substance, upon which the enzyme acts, is a tyrosine derivative (DOPA) believed to be formed in the adrenals. Melanin sythesis from the oxidation of phenylalanine or tyrosine are as follow:

1. Tyrosine → DOPA → DOPA → Quinone

2. DOPA - Quinone →2 - Carboxy 2,3 - dihydro - 5,6 - dihyroxyindole

→2- Carboxy - 2,3 - dihydro - indole - 5,6 - Quinone →5,6Dihydroxyindole.

3.5.6 Dihydroxyindole →Indole - 5,6 Quinone →Melanin

Melanin formation in both human and amphibian skin is augmented by the harmone known as intermedian or melanocyte - stimulating harmone (MSH) secreted by the pars intermedia of the pituitary gland. Adrenocartico tropic harmone (ACTH) secreted by Anterior Pituitary has melanocyte - stimulating activity similar to MSH although to a much lower degree. In addison's disease ACTH is secreted in a large amount and there is brownish black pigmentation of the exposed parts of the skin eg. hands, feet and mucous membrane.

Melatonin extract from bovine pineal gland, causes concentration of meanin near the nuclei of melanocytes in frog and as a result of this the skin becomes pale. Its role in the human is not

known. MSH causes the serum copper to rise and this is accompanied by in the melanin formation. Diminished formation of melanin is seen in albinism and leucoderms. In melanotic sarcoma, melanin may be found in the urine.

Aetiology - Vitiligo

Melanocytes in areas of depigmented skin are destroyed and the cause is unknown. Anti-melanocytic anti-bodies directed against intra cellular components of melnaocytes have been shown. The presence of organ specific auto immune disease occurs in about 10 % of patients. Such conditions are more common in their families than in a normal population. A neurogenic defect has been postulated for the rare dermatomal pattern of vitiligo which affects principally the limbs.

Endocrines - Association with throtoxicosis and diabetes.

Trophoneurosis and autonomic imbalance - emotional stress and strain.

Infections and toxic products, Enteric fever ill health, focal sepsis.

Drugs and chemicals - like quinines, guano furacin, amylphenol, chlorthiazide, broad spectrum antibiotics and chloroquin.

Auto-immune thyroid disease is one of a group of organ specific auto immune diseases that include pernicious anaemia, Addison's disease and hypo para - thyroidism.

Hereditary Factors

Hereditary is one of the factors supposed to be related with venpadai to some extent.

Familal incidence has been reported in 7.5 to 21 % in India and 33 to 40 % in western countries.

It is every day knowledge and observation that emotional factors affect the skin as shown by the blushing of embracement, the pallor of fear and the pallor or redness of Change, depending on the subject and his emotional state. Experiments have demonstrated that emotional change can affect the following, which has direct relevance in the aetiology of certain skin disorders.

- Control of vascularity of the skin
- Control of sebaceous gland secreation.
- Influencing the degree of oxidation.
- Influencing the tendency of pruritus.

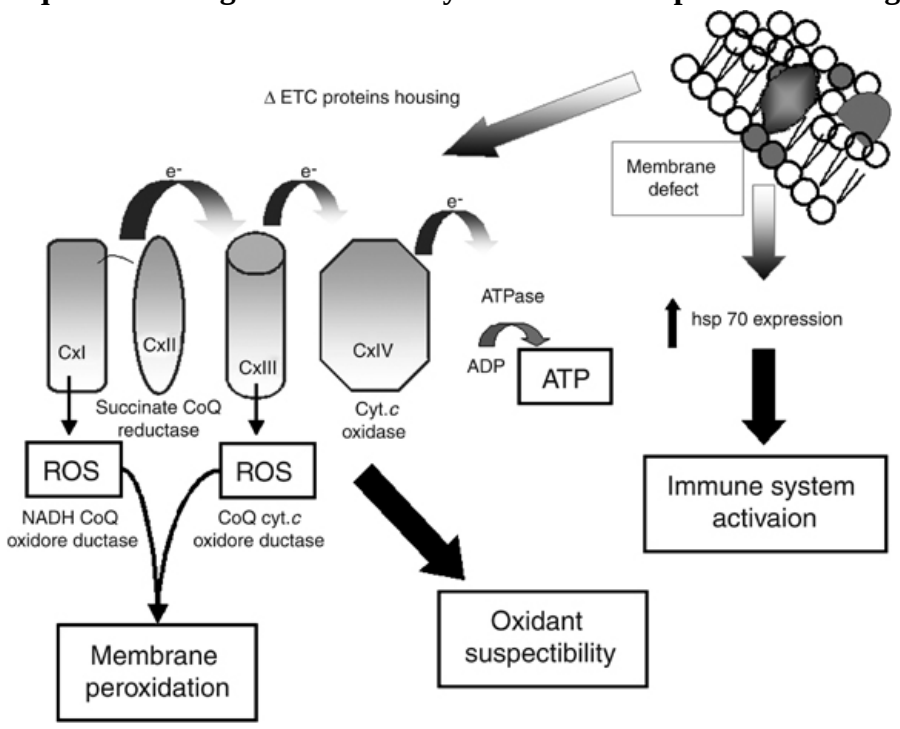
There is due to the causative factor of this disease, vanpadai from the following basic facts, it is generally considered to be a trophoneurosis. Phychological factors are known to be responsible for the pecipitation and aggravation of the disease.

Pathology

Chemically melanin pigment is a group of chromo proteins with coloured prosthetic groups, which is derived from the precursor tyrosine in the following way Tyrosine Tyrinase Dihydroxy phenylanin (DOPA) Melanogenase Melanin (Dopa oxidase).

Melanin + Protein = Melano protein

The possible pathogenetic mechanism. a schematic picture of the causal and temporal sequence leading to the melanocyte functional impairment during vitiligo.



In the skin, the pigment is produced by the melanocytes of their precursors melanoblasts. The melanoblasts are supposed to be derived from the cells of neuro ectodermal origin during the embryonic life. After birth, these cells migrate to their definitive position. The melanocytes appear as clear cells within the basal cell layer of the epidermis and show dendritic processes after special staining. These processes come in contact with similiar process of other melanocytes and epithelial cells through which the melanin pigments are donated to the basal

cells of epidermis. The dermis of normal skin also shows macrophages containing malanin pigments known as melanophores, which are incapable to produce the melanin pigments.

Causes of Hypopigmentation

Generalised depigmentation is found mostly in albinos. In this case, the characteristic dendritic melanocytes are present in the skin, but they are unable to produce melanin pigment due to defective tyrosinase activity. In albinism, the skin looks milky white, the hairs are pale looking and the iris is transparent. The generalized pallor is also noticed in panhypopituitiarism, male eunuchoidism and phenyl ketonuria.

Localised depigmentation is often noticed in the skin of patterned leucoderma. The white patches on the skin may be quite extensive and the condition is inherited as an autosomal dominant character.

Sometimes sharply defined focal depigmented areas are found on skin of persons suffering from vitiligo. In the affected areas, melanocytes are absent and there is no trace of melanin. The condition is an acquired one and shows some familial tendency.

Vitiligo in patients in whom the disease spreads very fast or those having halo-navi or malignant melanoma is believed to be based on auto-immune mechanisms, where auto antibodies or sensitized lymphocytes are supposed to act on the melanocytes. Trauma on the skin including that produced by scratching can lead to depigmentation of the skin even when it does not lead to ulceration. Leucoderma is also commonly seen on the flanks of ladies wearing tight petticoat strings where the prolonged pressure is presumed to lead to depigmentation. Sometimes vitiligo can be caused by the action of monobenzyle either on hydroquinone present in the slippers, gloves (or) other articles made of rubber or used as a depigmenting agent in the form of an irritant for pigmentary disorders. Recently vitiligo has also been observed to occur from plastic slippers as well as plastic ‘hindis’.

However most people with vitiligo have no other autoimmune disease. Vitiligo may also be herediatary, that is, it can run in families. Children whose parents have the disorder are more likely to develop vitiligo. However, most children will not get vitiligo even if a parent has it, and most people with vitiligo do not have a family history of the disorder.

Clinical features :

1) In this condition patches of skin lose their pigment and become perfectly white, though no other changes take place in them and particularly there is no scaling.

2) Vitiligo may occur in either sex and at any

3) The white patches may appear on any part of the skin but commonest on the face, neck, hands and wrist, lower abdomen and thighs and may be precipitates by trauma to the skin.

4) They may be of any size or shape and are usually though not always, roughly symmetrical.

5) They slowly increase in size until large areas of the skin are completely discoloured.

6) The remaining small patches of normal skin may then be mistaken for pigmented areas. The mistake may be avoided by remembering that the vitiligo areas have convex margins and the normal areas therefore have concave ones.

7) When vitiligo occurs on a hairy area such as eyebrows or pubis the hair on the white patch may also become white.

8) The depigmented areas are sometimes surrounded by an excess of pigmentation in the immediately adjoining skin but this appearance is often illusory and the result of visual contrast.

9) Vitiligo is most noticeable in the summer when the normal skin is tanned by the sun.

10) Vitiligo sometimes disappears spontaneously after months or years but more usually the conditions spreads slowly and may eventually involve nearly whole of the skin.

11) It is characterized by completely depigmented macules and patches of varying sizes and shapes.

12) There are no other changes except depigmentation.

13) Early lesions may be pale white and ill defined. At this stage, Wood's lamp helps to confirm the diagnosis. Patches enlarge slowly and may affect the whole body.

14) Any part of the body can be affected but the sites of predilection are the face, dorsa of fingers and hands, wrist and the legs.

15) Involvement of mucous membrane especially the lip is not uncommon; it can precede cutaneous involvement by years.

Clinical Criteria for Classification of Vitiligo

Stages of Clinical Features

Vitiligo

Active (VI)	(i) New lesions developing
	(ii) Lesions increasing in size
	(iii) Border ill-defined
Quiescent (V2)	(i) No new lesions developing
	(ii) Lesions stationary in size
	(iii) Border hyper pigmented and well-defined.
Improving (V3)	(i) Lesions decreasing in size
	(ii) No new lesions developing
	(iii) Border defined and signs of spontaneous regimentation

Zosteriform: Unilateral distribution of lesions, preferably along the course of nerves.

Besides typing the stage of disease, it is useful to decide the variety (acral,Vulgaris, Zosteriform). Severity (Localized or extensive) and acuity (insidious or galloping) of vitiligo.

Diagnosis :

- The distribution, the age of onset and the hyperpigmented border will suggest the diagnosis.
- It is usually apparent; in doubtful and early case, Wood's lamp is of great help in diagnosis.
- Usually in macular leprosy, seborrhoeides, pityriasis versicolor and nevoid condition, its assistance is called for.

- In piebaldism the lesions are present at birth, are usually confined to the head and trunk and rarely show a hyperpigmented border.
- Careful examination of the texture of the unpigmented skin should exclude lichen sclerosus and scleroderma.
- Post-inflammatory leucoderma, which is frequent in the darker races, shows an irregular mottling of hyperpigmented and hypopigmented blotches.
- Hypomelanosis of the affected skin is commonly seen in pityriasis alba, producing slighlty scaly areas with rather ill defined edges of children's faces.
- Hypopigmented, slighly scaly macules are seen in pityriasis versicolor.
- Vitiliginous areas are milky white while other lacks this milky white colouration.
- Stationary patches are well-defined and have hyperpigmented borders.
- Absence of scaling, crusting and itching help to eliminate seborrhoeids and pityriasis versicolor.
- These areas often fluorescence a golder yellow when examined under a Wood's lamp. The hypomelanotic macules in leprosy are anaesthetic.
- Examination of the skin in long wave UVR helps distinguish whether there is total depigmentation (as in Vitiligo) or not. It may also detect areas of depigmentation not easily seen in ordinary daylight, as well as detecting a lemon-yellow fluorescence seen in some cases of pityriasis versicolor.

Prognosis

It has improved considerably in recent years because of better understanding of etiological factors and advances made in therapy.

Following conditions are said to be of poor prognosis.

1) Poor nutritional state or digestion, use of broad - spectrum antibiotics over long period. Emotional stress and nervous debility.

2) Presence of vitiligo on resistant sites like the hands and the feet, front of wrists, the elbow, the waist, the eyelids and lips.

3) Depigmented hair in vitiliginous areas.

Causes of Localised Hypopigmentation

Vitiligo	Destruction of melanocytes; common; acquired, multiple sharply defined nonpigmented patches any where.
Pityriasis versicolor	Superficial fungus infection leading to disturbance in pigment production, common multiple pale scaling patches on trunk
Pityrisis alba	Mild patchy eczema of the face in children causing a disturbance in pigment production.
Leprosy	One or several paler macules on trunk or limbs that are hypoaesthetic.
White macules of affecting tuberous sclerosis	Uncomming development of anomaly of CNS, connective tissue and skin; several “maple leaf” shaped hypopigmented macules.
Postinflammatory hypopigmentation	After inflammatory skin disease (after eczema or trauma to the skin; irregular in shape and in depth of pallor).
Naevous anaemicus	Rare developmental solitary white patch uaually on trunk; thought to have vascular basis.
Chemical toxicity	May look very much like vitiligo; seen in workers in rubber industry exposed to parateriary benzyltoluence.

Differential Diagnosis of the important Depigmentary Disorders

Distinguish Features	Albinism	Naevus Depigmentosus	Vitiligo	Leprosy	Pityriasis
Age	Congential present at birth	Congential-present at birth	Acquired	Any age	Any age
Distribution	Complete (or) partial	Unilateral	Any area	Any area	Trunk, Neck, and Face
Course	Stationary	Does not increase in size or changing shape	Progressive	Progressive	Progressive;worse in mansoon and summer
Hyperpig mentary border	Nil	Nil	Present	Inflammatory	Nil
Heredofamilial	Hereditary	Not herediotry	Nil	Nil	Nil
Other features	Hair and eyes may be effected	Nil	Nil	Anethesia thickened nerves, nasal, bleeding slit smear and biopsy	Furfuraceous like dandruff, scaling in head macules and large patches.

5.RESULTS AND OBSERVATIONS

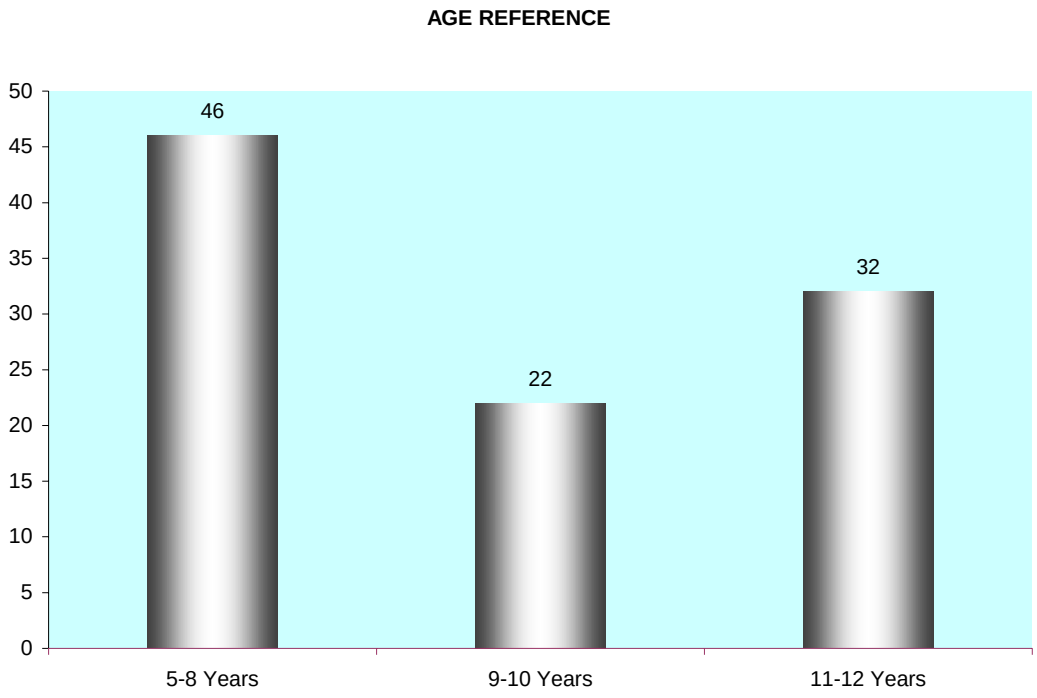
Results were observed with respect to the following criteria

1. Age reference
2. Sex reference
3. Religion reference
4. Socio Economic status of the reference
5. Etiology reference
6. Family history reference
7. Paruva kaalam
8. Diet reference
9. Thinai
10. Site of lesion
11. Derangement in the types of Vatham
12. Derangement in the types of Pitham
13. Derangement in the types of Kabam
14. Ezhu Udar Kattugal reference
15. Ennvagai Thervugal reference
16. Distribution of nadi among the patient with venpadai
17. Neikuri reference
18. Results after treatment reference

The observations recorded with the above said criteria are given in the tabular form.

1. Age Reference

Age (in Years)	No. of Cases 50	Percentage %
5-8 Years	23	46
9-10 Years	11	22
11-12 Years	16	32

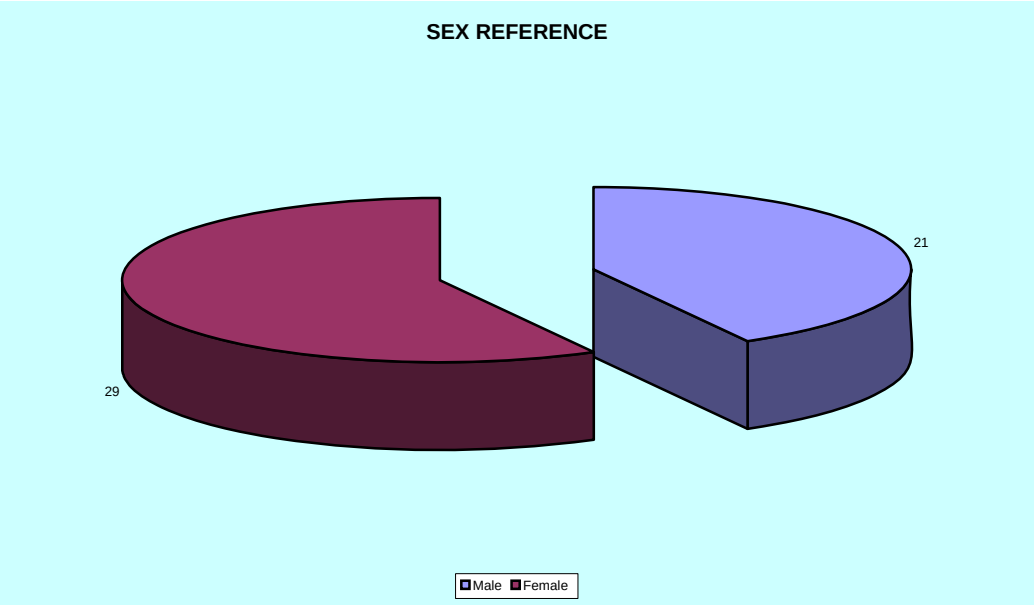


Inference

About 46% of them were aged 5-8 years and 32% cases 11-12 years, 22% cases 9-10 years.

2. Sex Reference

Sex	No. of Cases (Out of 50)	Percentage %
Male Children	21	42
Female Children	29	58



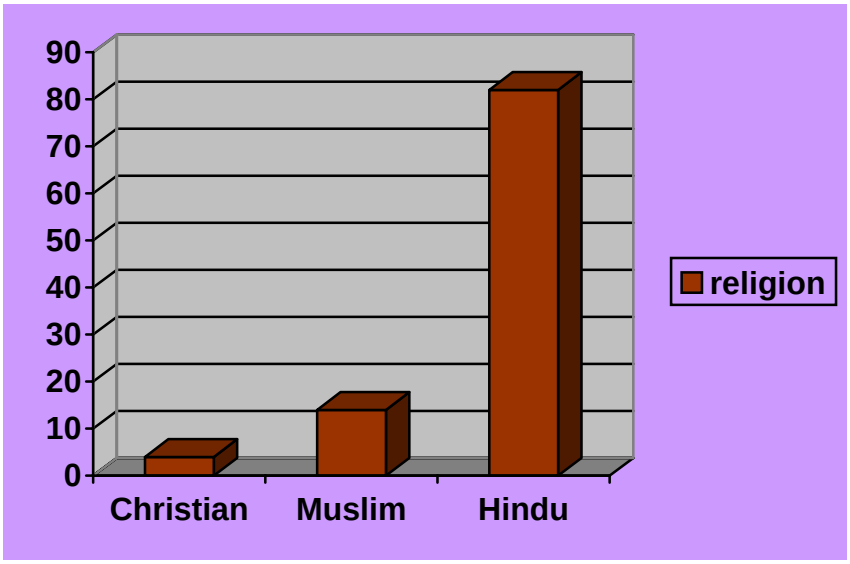
Inference

Out of 29 patients 58% were Female Children and 21 patients 42% were Male children.

3. Religion Reference

Religion	No. of Cases out of 50	Percentage %
Hindu	41	82
Christian	2	4
Muslim	7	14

Religion reference

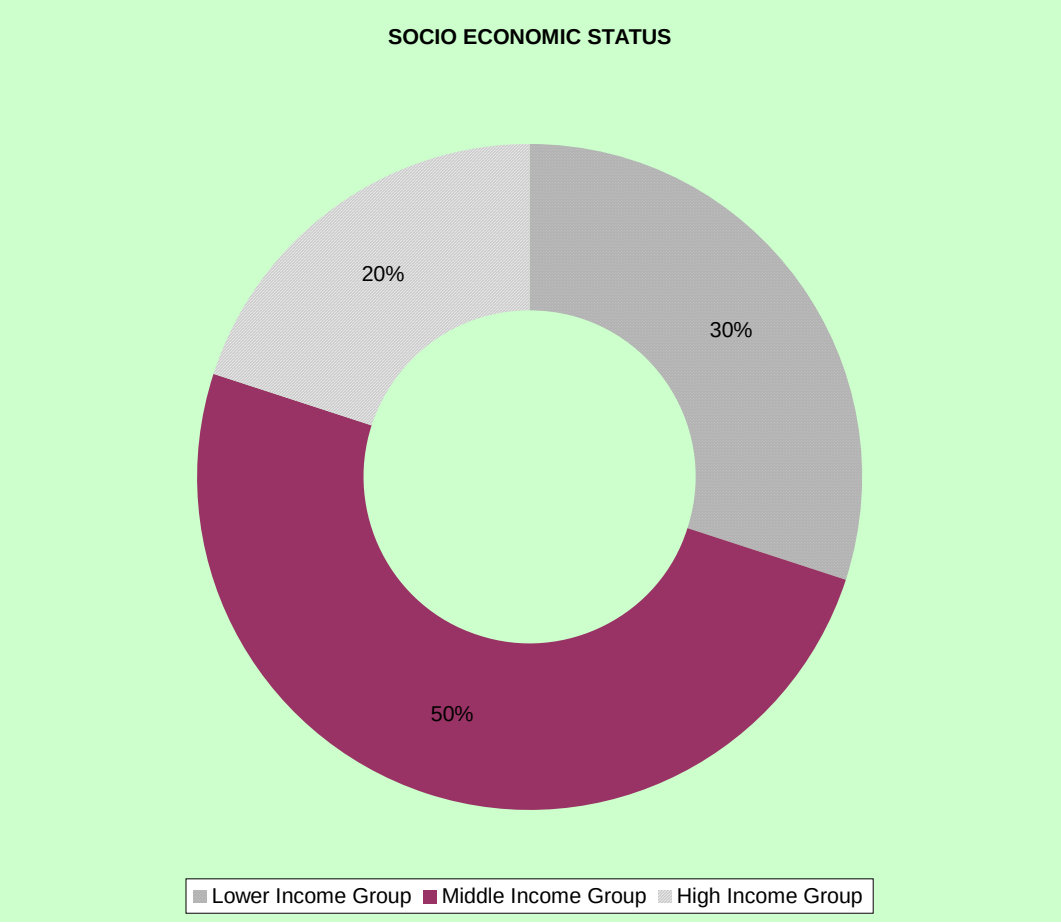


Inference

Most of the patients religion Hindu (82%) and Muslims 14% Christians 4%.

4. Socio – Economic Status

Socio – Economic Status	No. of Cases out of 50	Percentage %
Lower Income Group	15	30
Middle Income Group	25	50
High Income Group	10	20

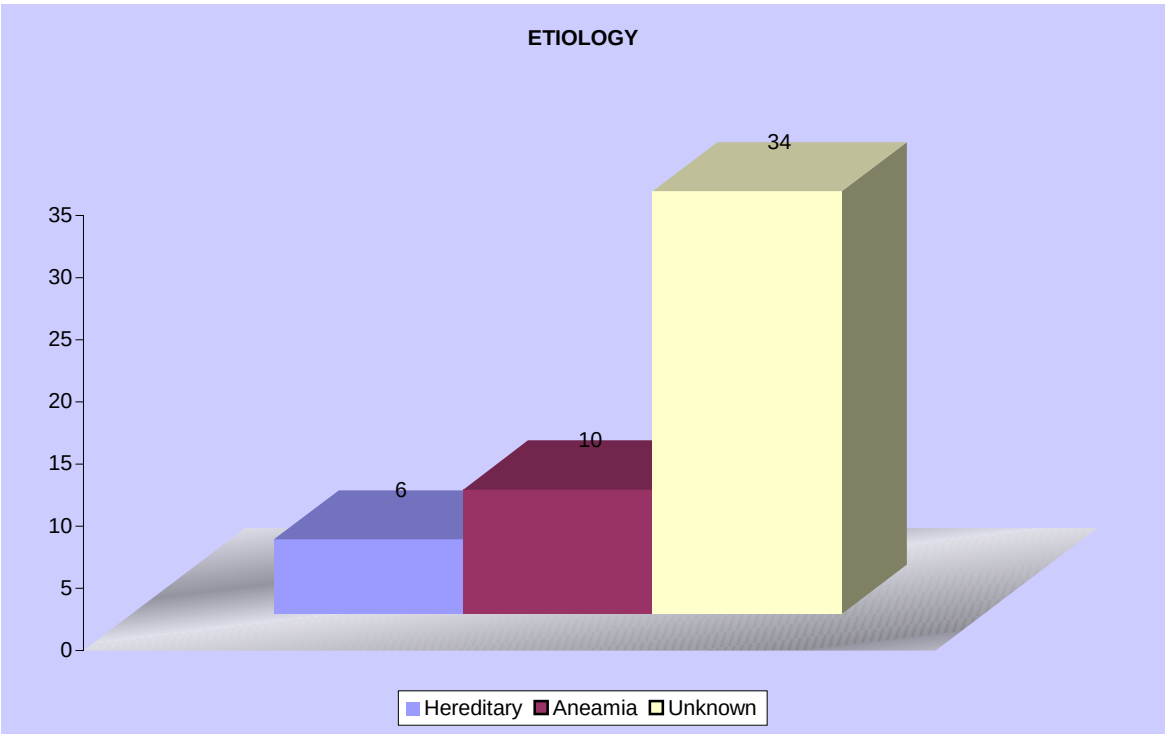


Inference

About 50% of the parents were middle income group and 15% lower, 20% high income group.

5. Etiology Reference

Etiology	No. of Cases out of 50	Percentage %
Hereditary	6	12
Aneamia	10	20
Unknown	34	68

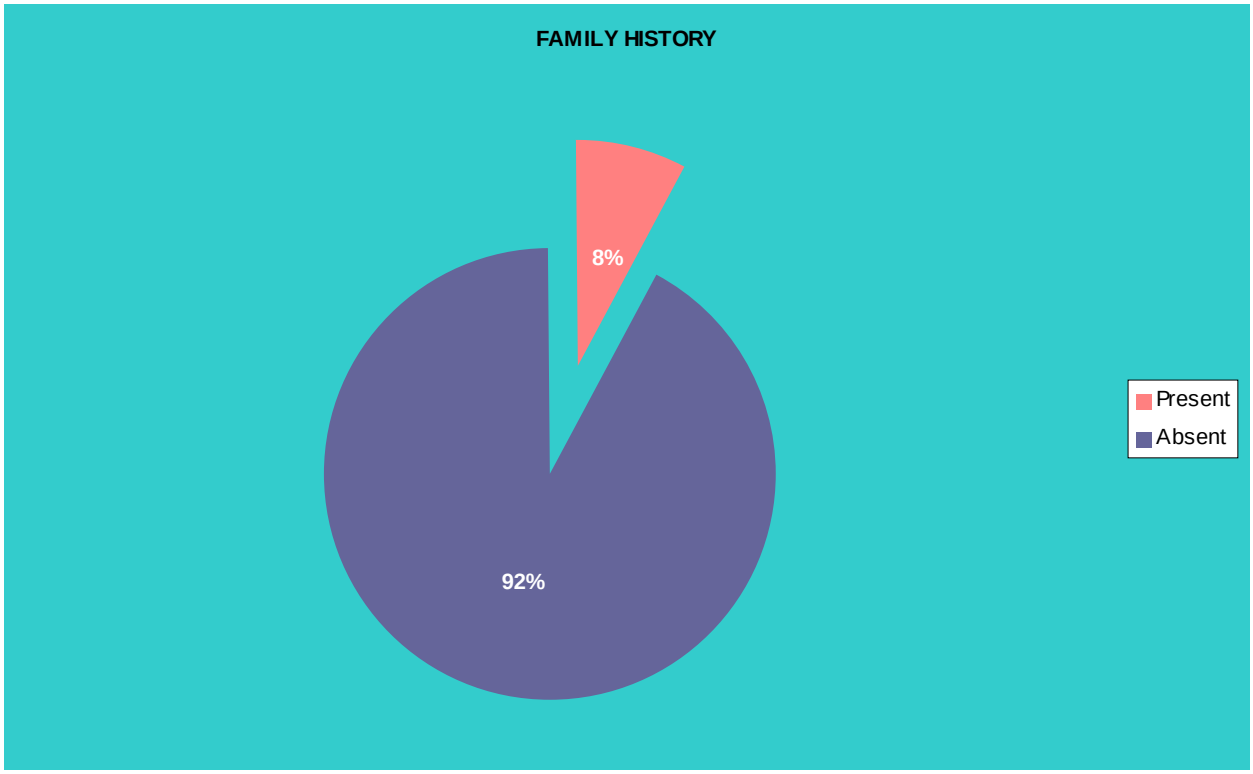


Inference

About 68% of the cases were of unknown aetiology and 20% Aneamia cases, 12% hereditary cases.

6. Family History Refernce

Family History	No. of Cases out of 50	Percentage %
Present	4	8
Absent	46	92

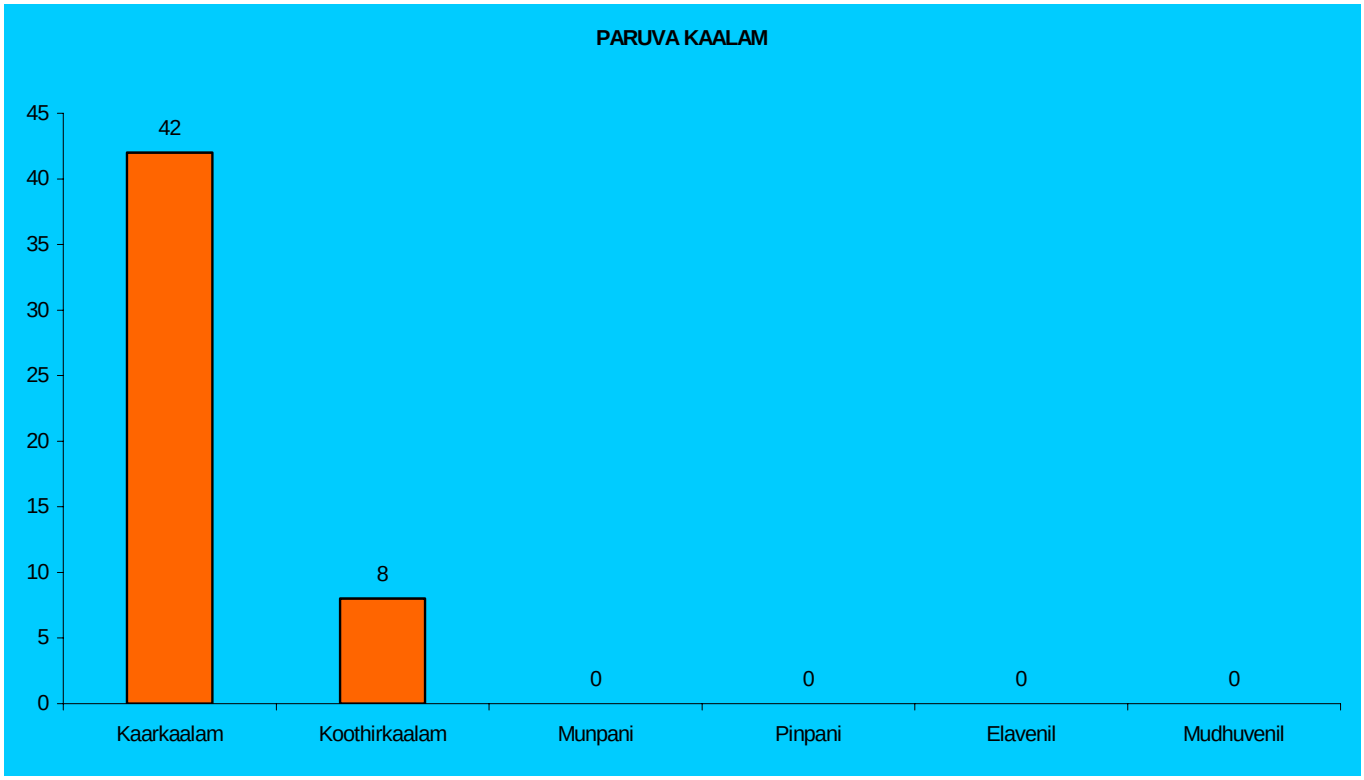


Inference

About 92% cases were having no family history absent and in 8% of cases family history was present.

7. Paruvakaalam

Paruvakaalam	No. of Casesout of 50	Percentage %
Kaarkaalam	42	84
Koothirkaalam	8	16
Munpani	0	0
Pinpani	0	0
Elavenil	0	0
Mudhuvenil	0	0



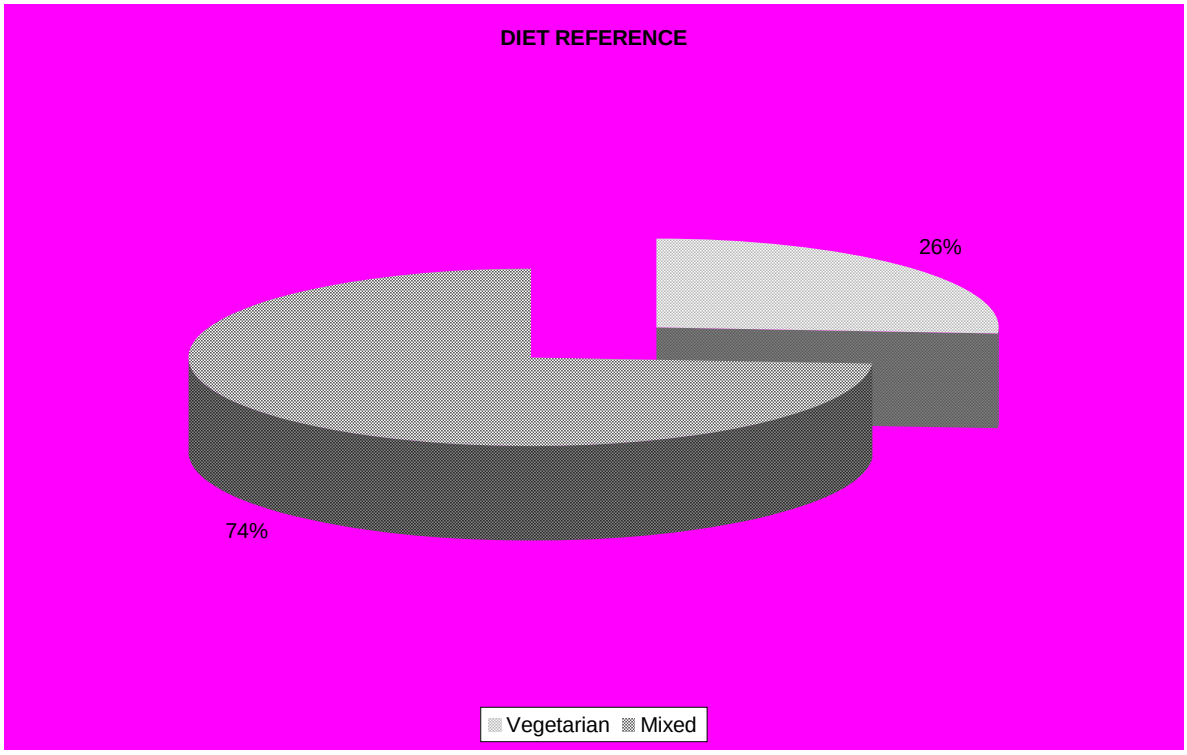
Inference

According to Paruvakkalam, highest incidence 84% were noted in Karkaalam and Koothirkalam 16% cases were noted (Table 7)

Out of fifty cases, 42 were admitted in Kaarkaalam and 8 admitted in Koothirkaalam and none in the remaining kaalangal.

8. Diet Reference

Food Habit	No. of Cases out of 50	Percentage %
Vegetarian	13	26
Mixed	37	74

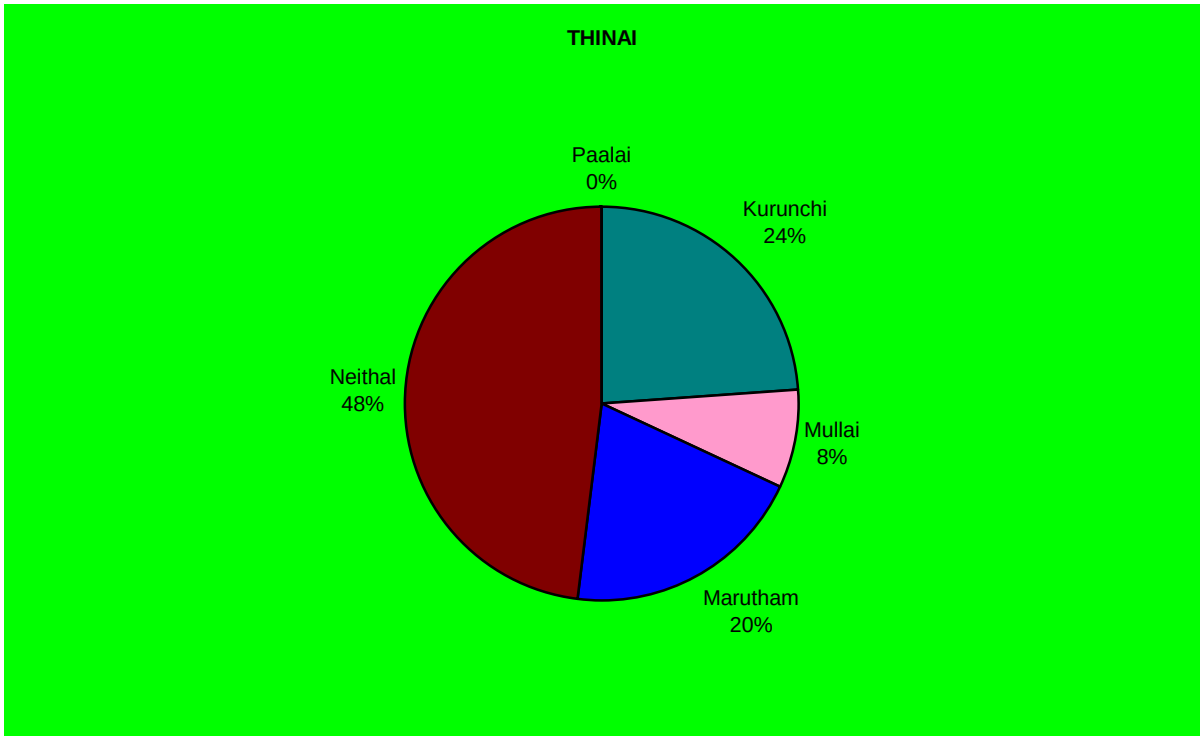


Inference

About 74% of cases were taken mixed diet and 26% of cases have Vegetarian.

9. Thinai

Thinai	**No. of Casesout of 50**	**Percentage %**
Kurunchi	12	24
Mullai	4	8
Marutham	10	20
Neithal	24	48
Paalai	0	0

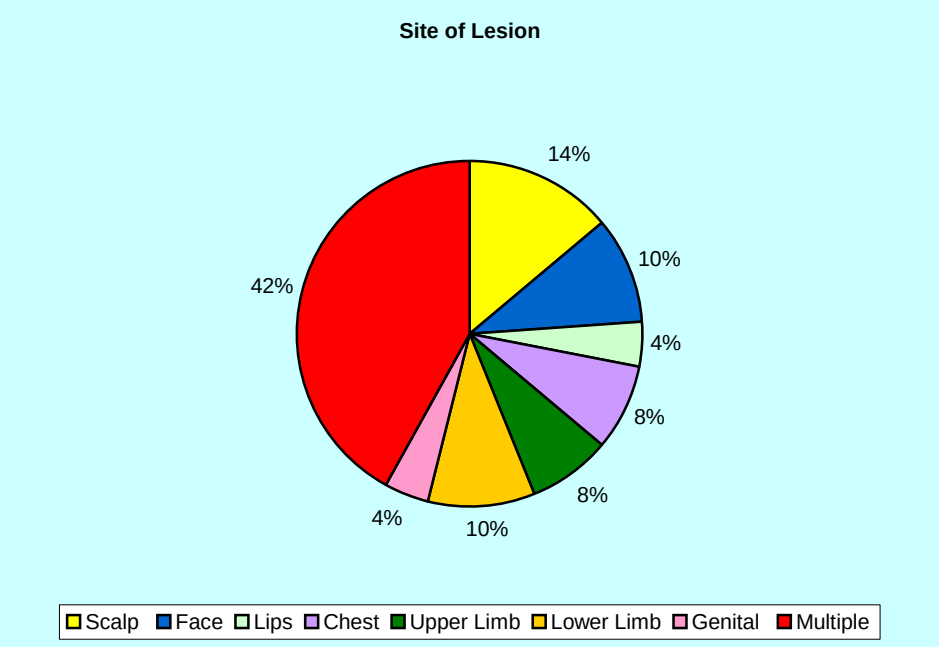


Inference

According to Thinai the highest distribution 48% was noted in Neithal, In Kurinchi 24%, in Marutham 20% and Mullai 8% were observed (Table 9).

10. Site of Lesion

Site of Lesion	No. of Cases out of 50	Percentage %
Scalp	7	14
Face	5	10
Lips	2	4
Chest	4	8
Upper Limb	4	8
Lower Limb	5	10
Genital	2	4
Multiple	21	42

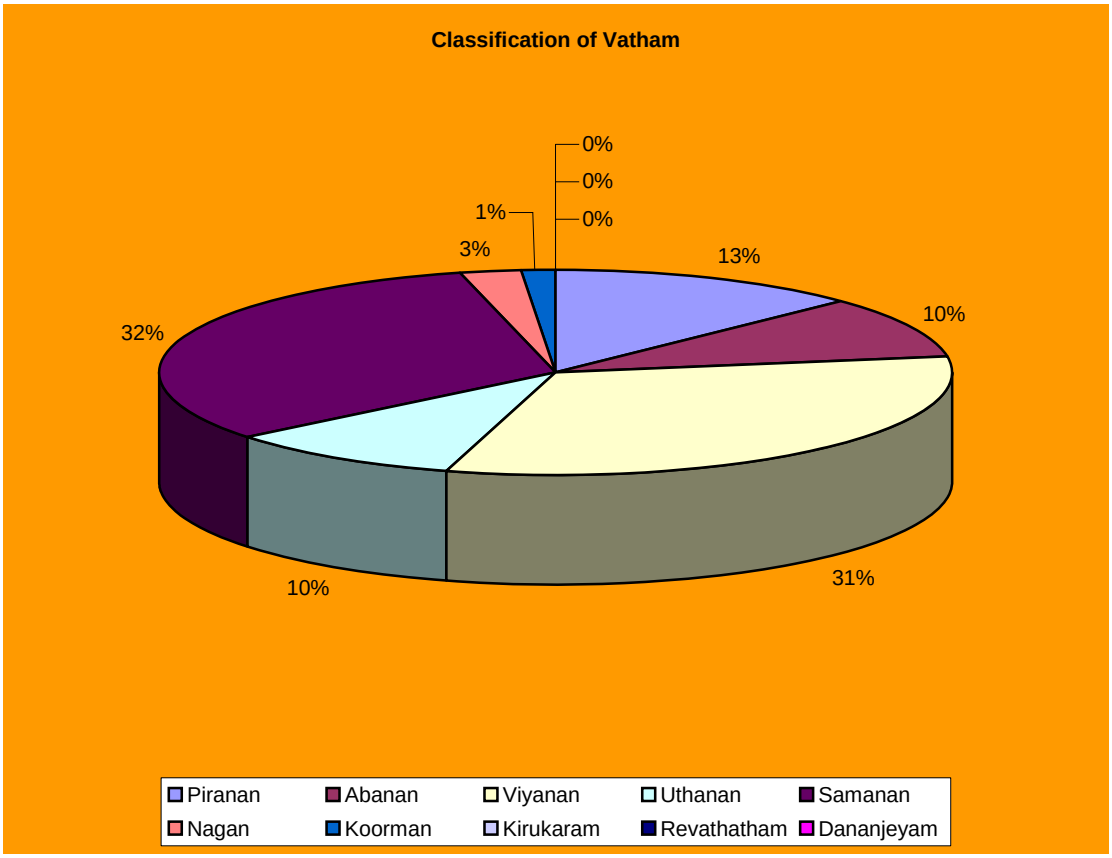


Inference

About 42% of cases were having multiple site of lesion, Scalp region 14%, Face 10% Chest 8%, upper limb 8% Lower limb 10% lips 4%, Gential region 4%.

11. Derangement in the types of vatham

Classification of Vatham	No. of Cases (out of 50)	Percentage %
Piranan	20	40
Abanan	15	30
Viyanan	50	100
Uthanan	15	30
Samanan	50	100
Nagan	4	8
Koorman	2	4
Kirukaram	0	0
Revathatham	0	0
Dananjeyam	0	0

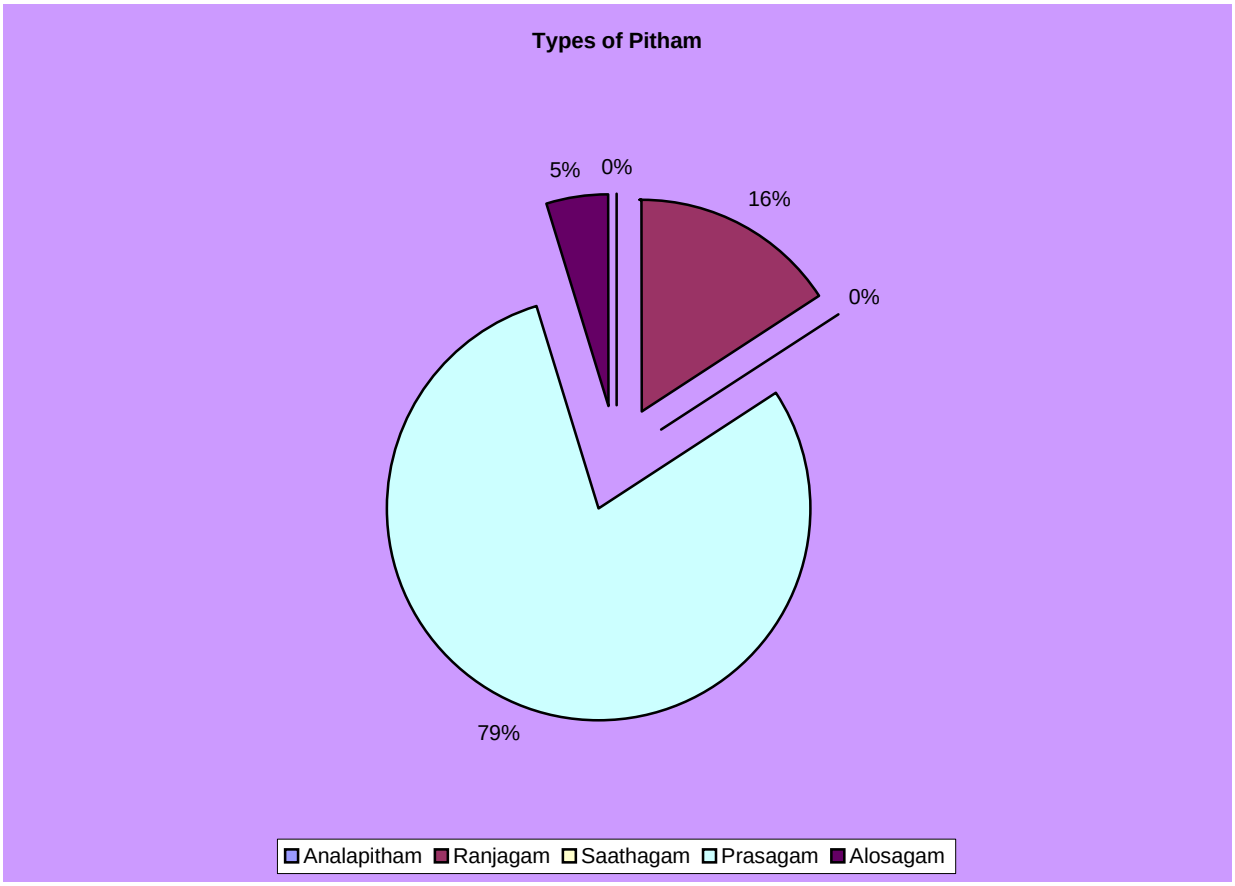


Inference

According to classification Vatham derangement of Viyanan was 100%, Samanan was 100% Piranan was 40%, Uthanan was deranged in 30% Nagan 8% Koorman 4%.

12. Derangement in Types of Pitham

Types of Pitham	No. of Cases out of 50	Percentage %
Analapitham	0	0
Ranjagam	10	20
Saathagam	0	0
Prasagam	50	100
Alosagam	3	6

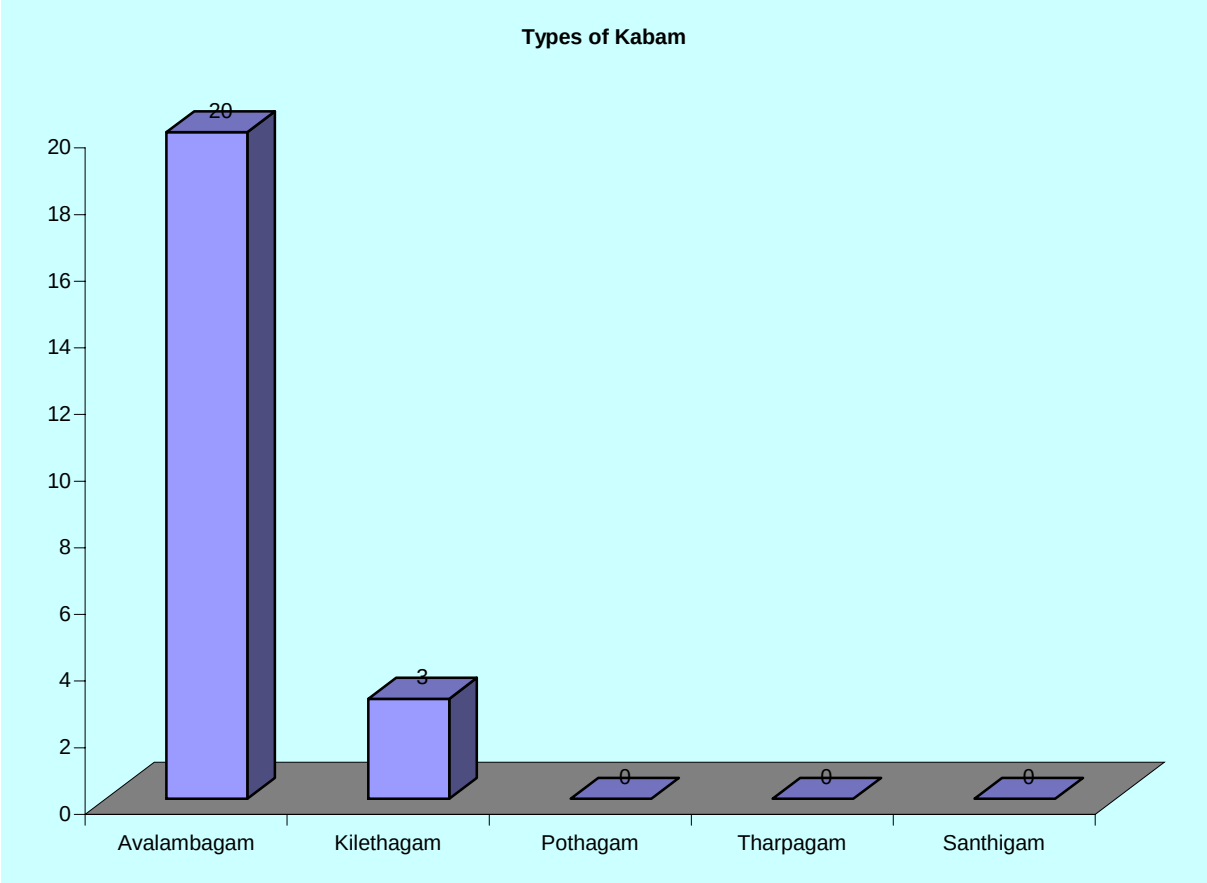


Inference

According to Pitham Prasayam (Oollolither 100%) was affected, Ranjagam (Varma eri) was affected in 20% Alosagam (Nokanal) was affected in 10%.

13. Derangement Types of Kabam

Types of Kabam	No. of Cases out of 50	Percentage %
Avalambagam	20	40
Kilethagam	3	6
Pothagam	0	0
Tharpagam	0	0
Santhigam	0	0

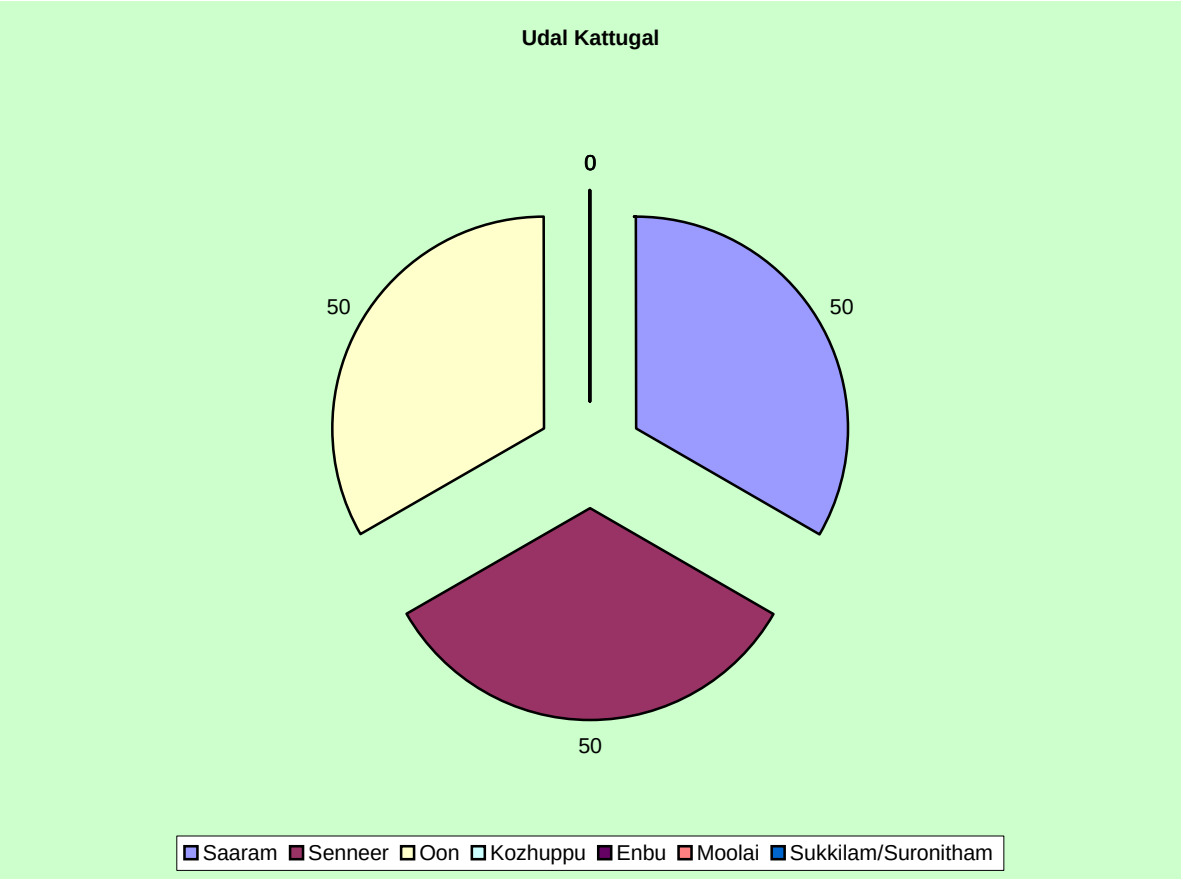


Inference

According to Kabam Avalambagam was deranged in 40%, Kilathagam was affected in 6% Pothagam was affected in 16% Pothagam was affected in 16% of cases.

14. Ezhu Udal Kattugal reference

Udal Kattugal	No. of Casesout of 50	Percentage %
Saaram	50	100
Senneer	50	100
Oon	50	100
Kozhuppu	0	0
Enbu	0	0
Moolai	0	0
Sukkilam/Suronitham	0	0

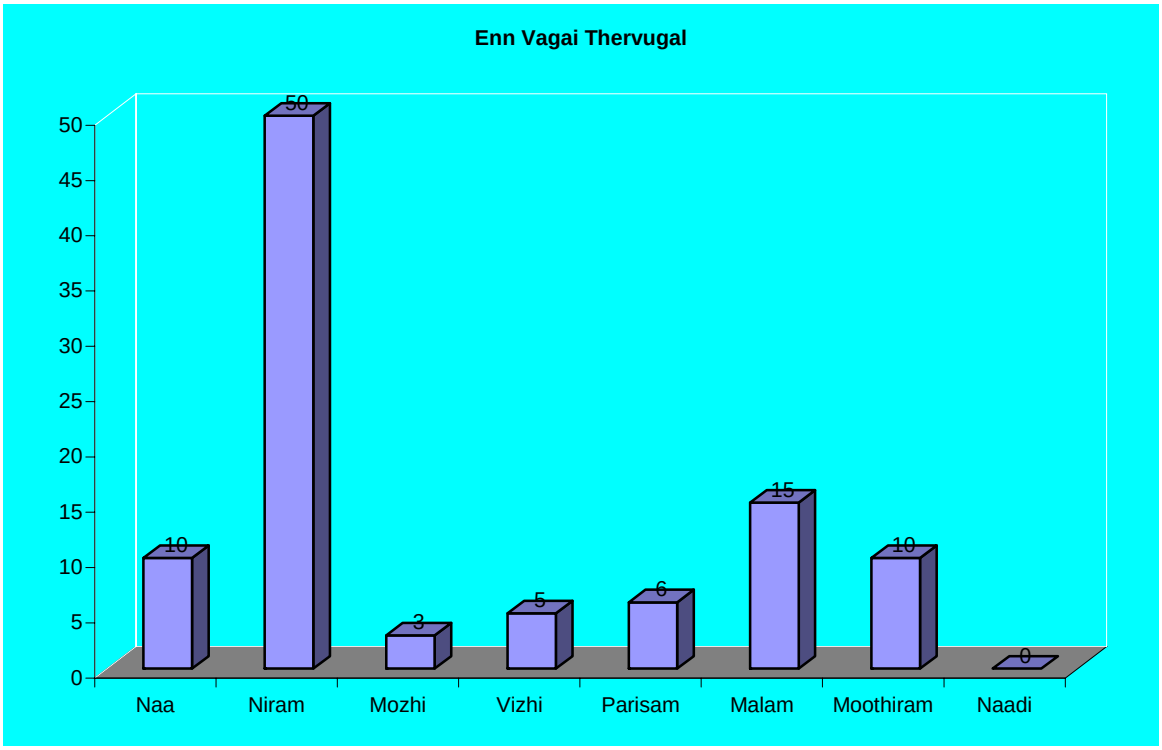


Inference

Saaram was affected in 100% of cases Senneer was affected in 100% of cases Oon was affected in 100% cases.

15 Enn Vagai Thervugal Reference

Enn Vagai Thervugal	No. of Casesout of 50	Percentage %
Naa	10	20
Niram	50	100
Mozhi	3	6
Vizhi	5	10
Parisam	6	12
Malam	15	30
Moothiram	10	20

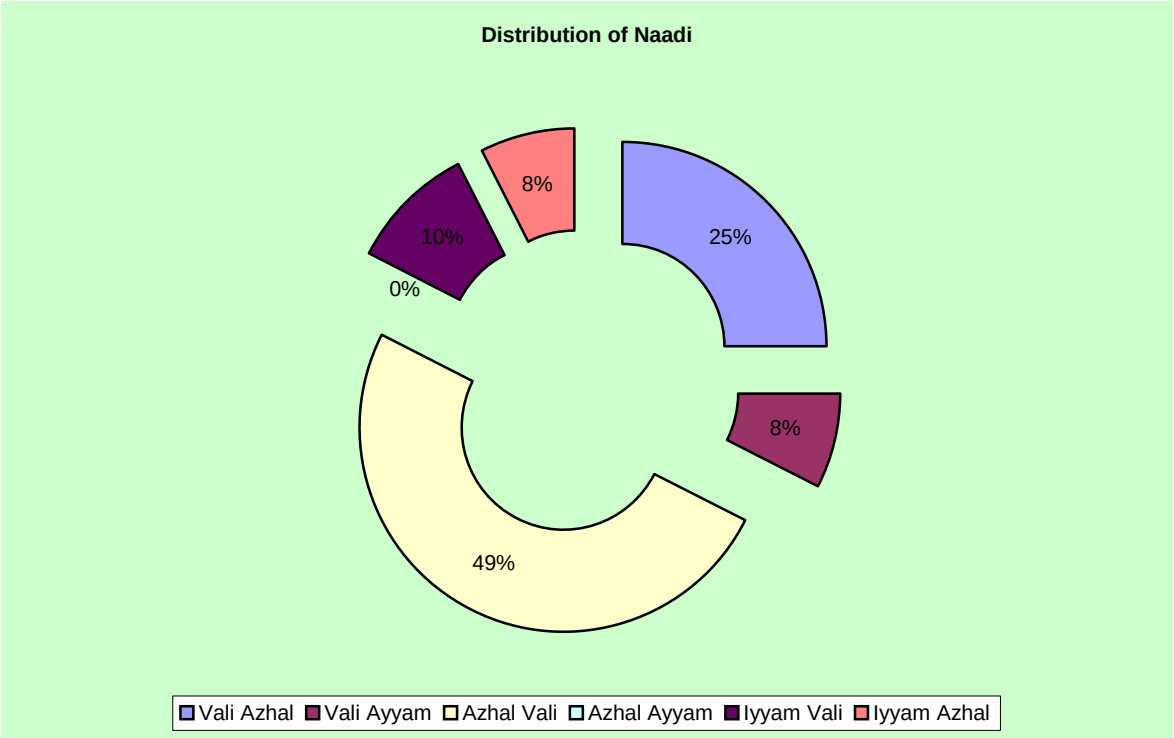


Inference

Niram was affected in 100% of cases and Malam was affected in 30%, Moothiram 20% Naadi 20%, Sparisam 12% Vizhi 10% and Mozhi 6% of cases.

16. Distribution of nadi among the patient with venpadai

Nadi	No. of Casesout of 50	Percentage %
Vali Azhal	10	20
Vali Ayyam	3	6
Azhal Vali	20	40
Azhal Ayyam	0	0
Iyyam Vali	14	28
Iyyam Azhal	3	6



Inference

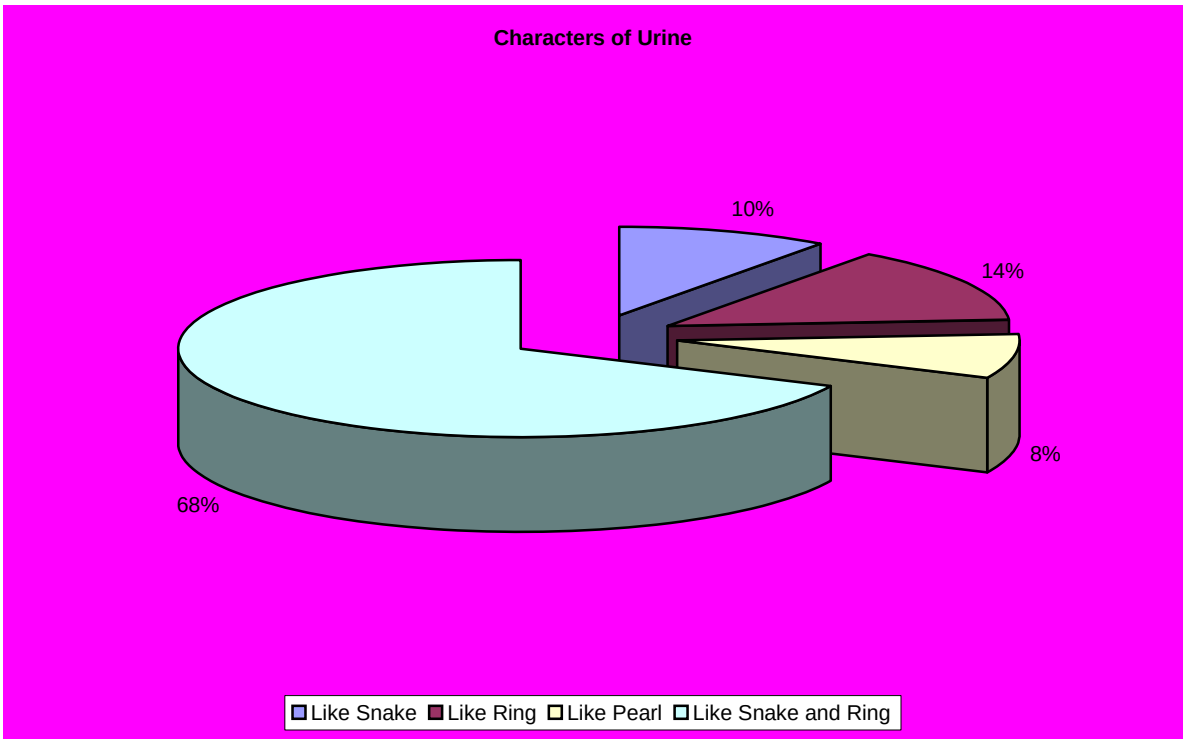
Azhal vali was observed in 40%

Azhal ayyam was observed in 20 %

Vali Azhal 20%, Ayam vali 28%, Ayam azhal 6%, Vli ayam 6%

17. Neikuri Reference

Character of Urine	Neikuri Reference	No. of Cases out of 50	Percentage %
Spreads Like Snake	Vatha Neer	5	10
Spreads Like Ring	Pitha Neer	7	14
Spreads Like Pearl	Kaba Neer	4	8
Spreads Like Snake and Ring	Thontha Neer	34	68

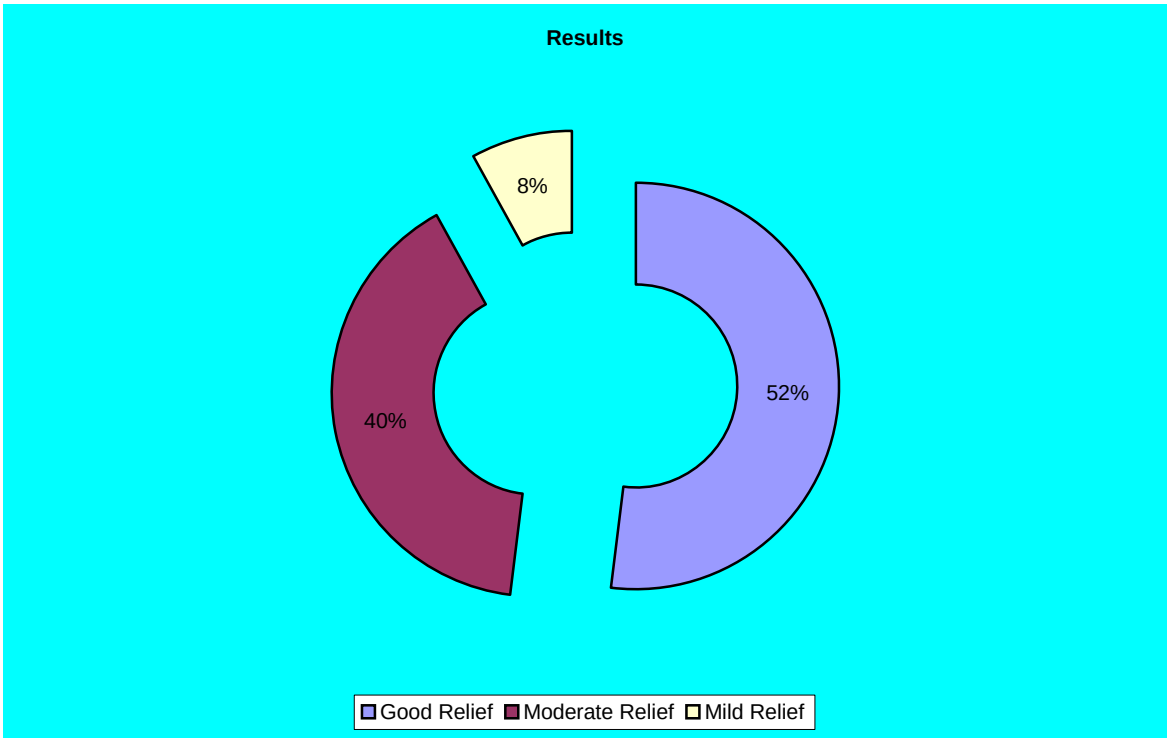


Inference

Thontha Neer affected in 68% of cases Pitha neef 14% vatha neer 10% and Kaba neer 8%.

18. Results

Results	No. of Cases out of 50	Percentage %
Good Relief	26	52
Moderate Relief	20	40
Mild Relief	4	8



Inference

About 52% of cases had good relief and 40% had moderate relief and 8% mild relief.

6.DISCUSSION

According to Siddha literature Venpadai is one of the eighteen types of kuttam. The clinical entity of venpadai is more or less similar to that of vitiligo in modern medicine. It is an acquired idiopathic depigmentary condition and is characterized by completely depigmented macules and patches of varying sizes and shapes. Beside loss of colour there is no other structural change.

Anatomy and physiology of skin and aetiology, clinical feature of the disease etc., are discussed. Author collected these largely from kuzhanthai Maruthuvam and Siddha methods of diagnosis was carried out. Before and after the course of treatment patients were subjected to laboratory investigations and photographs were taken.

Laboratory Investigation :

Blood – TC, DC, ESR, Hb, Urea and Serum cholesterol

Urine – Albumin, Sugar and deposit

Neerkkuri and Neikkuri

Aetiology

The causative factors of the disease can be found out from the history given by the patients, their diet, habits, occupation, mental stress and other signs and symptoms. According to the Siddha and modern science the aetiology of the disease has been arrived at as follows :

- Nutritional deficiency of copper, protein, vitamins in diet
- Gastro-intestinal problems like amoebiasis, helminthiasis, chronic diarrhoea and dysentery.
- Hereditary and autoimmune disorders.
- Thyrotoxicosis and diabetes.
- Industrial chemicals and dyes.
- Infections and toxic products, enteric fever, ill health, focal sepsis.
- Chronic irritation
- Anaemia
- Unknown aetiology

Gender Distribution:

50 patients of both the gender were selected for the dissertation study. Among the 50cases 21 (42 %) were males and 29(58%) were females. The gender distribution was more or less equal.

Age Distribution:

23(46%) Patients were in the age group between 5-8

11 (22%) patients were in the age group between 9-10

16 (32%) patients were in the age group between 11-12

Dietary Habits:

37 (74%) patients were non vegetarians and only 13 (26 %) were vegetarian

Seasonal variation:

42 (82 %) cases were admitted to trial in kar kaalam and 8 (16 %) koothir kaalam.

Thinai:

12 (24 %) patients belonged to kurunchi thinai,

4(8 %) patients belonged to mullai thinai,

10(20 %) patients belonged to marutham thinai,

24 (48 %) patients belonged to Neithal thinai,

In Siddha literature the Marutham is mentioned as disease free land among the five lands. Because of various environmental changes in the life style, venpadai occurs irrespective of the land, Maximum patients came from in and around Chennai which belongs to Neithal thinai.

Reference of Mukkutram

1. Vaatham

piranan was affected in 20 (40 %),

Abaanan was affected in 15 (30 %),

Uthanan was affected in 15(30 %),

viyanan was affected in 50 (100 %),

samanan was affected in 50(100%),

nagan was affected in 4 (8%),

koorman was affected in 2 (4%)

koorman in 3 (7.5 %)

2. Pittham

Ranjagam was affected in 10(20%)patients,

praasakam was affected in 50(100 %) patients,

alosagam was affected in 3(6%) patients

Praasakam is responsible for the complexion of the skin. Due to defect in praasakam skin colour is changed in Venpadai.

3.Kabam

Avalambagam was affected 40 % Kilethagam was affected 6%

Udal Kattugal

Saaram, Senneer and oon which are responsible for the colour of the skin were affected in all the 50 (100 %) cases.

Ennvagai thervugal

Naa was affected in 10(20%) cases

Niram was affected in all the (100 %) cases because in venpadai, the colour of Skin changed into white.

Mozhi was affected in 3(6%) cases.

Vizhi was affected in 5 (10%)cases.

Sparisam was affected in 6 (12%) cases

Malam was affected in 15 (30 %) cases.

Moothiram was affected 15 (30%) cases

Management:

In Siddha System before starting the treatment it is necessary to bring the three thadu into equilibrium. To bring down the vitiated vaatham, Viresanam (purgation) with Poovarasampattai ennai –5 ml with hotwater was given in the early morning.

Drugs

I. Lagu seenachoora choornam – 1gm (BD) with hotwater. (Internal)

II. Venpadai pattru(venkutta lepanam) – with cows urine (External)

Patients were instructed to take the medicines regularly and apply the pattru twice a day and to expose the affected parts to sunlight. Diet restrictions were strictly imposed and followed.

The main reasons for choosing the drugs mentioned above as trial drugs are :

Venpadai is the disease that affects not only the skin (body) but also the mind. These patients face a lot of problems not only in their own families but also in the society. It leads them to mental depression and stress. It aggravates the clinical condition. The treatment already available for venpadai is a prolonged one and does not fully cure the condition. Resorting to plastic surgery proves costly and it is not affordable to all the patients. Moreover the relapse of the disease at the site of surgery is common.

The trial drug Lagu seenachoora choornam is the best combination of herbs. Each content of the Lagu seenachoora choornam is specifically given for skin diseases, as described in pothu gunam.

7.SUMMARY

- Venpadai has been chosen for the dissertation work by the author.
- Various literatures dealing with Venpadai have been collected from Siddha and modern text books.
- Preclinical analyses in toxicological and biochemical aspects were conducted for the trial drug ***Lagu seenachoora choornam***. 50 patients of both sex and in age group between 5 to 12 were selected for the study.
- 20 cases were treated in inpatient ward at least for 30 days and followed up in the out patient department after discharge. 30 cases were treated in the outpatient department for 48 days.
- All the details about the study and the drugs were informed to the patients and consent forms obtained from them. Before starting the treatment, the blood samples of the selected patients were subjected to investigation and photographs of the lesions were taken.
- A day before starting the treatment purgation was given by administering sithathi ennai 1ml with hot water in the early morning to bring the thridosha to equilibrium.
- From the second day onwards ***Lagu seenachoora choornam*** 1 gram BD along with ***Venpadai pattru (venkutta lepanam)*** – with cows urine for external use were given to the patients.
- Diet restrictions were strictly followed during the treatment period. Every 8^{th} day the patients were assessed for clinical improvement and adverse effects. On the 48^{th} day the laboratory investigations and photographs were repeated. The improvement was assessed.
- During the course of treatment there were no adverse effects or unwanted drug reactions like itching, abdominal discomfort, and nausea.

8.CONCLUSION

- Venpadai may occur due to various causes and it leads to mental stress and strain. Hence it is one of the Psychosomatic disorders. When the trial drug Lagu seena chooranam(Internal) with Venpadai pattru (venkutta lepanam - External) were administered to the venpadai patients, it showed improvement in varying degrees in all the cases.

- No adverse effects were noticed during treatment period. The ingredients are lagu seena chooranam are of plants origin, easily available and harmless to children. The cost comparatively very low the medicine has many properties to control the signs and symptoms of venpadai.

- Clinical results were found to be having good relief in 52% of cases, moderate relief 40% of cases, mild relief 8% of cases.

- Because of the encouraging result clinically the study may be undertaken with same drug for prolonged period of time in the large number of cases and it may throw new lights for the treatment of venpadai (vitiligo).

- The trial drug ‘lagu seena chooranam’ denoted in Agathiyar vaithya pillaitamil and venpadai pattru (External) denoted in pathartha guna villakam text as a common effective drug for venpadai and through this trial the effectiveness of lagu seena chooranam (Internal) and venpadai pattru (External) was conferred and re-established by the author.

1.PREPARATION AND PROPERTIES OF TRIAL DRUG

Trail Medicine I (Internal)

Name of the Medicine: இலகு சீன சூரணம்

Reference : அகத்தியர் வைத்திய பிள்ளைத் தமிழ்
பக்கம்- 72

சேரும் சரக்குகள்

சுத்தி செய்த பரங்கி பட்டை	7½ வராகன்
சிவனார் வெம்பு வேர்பட்டை	7½ வராகன்
சிறுகுறிஞ்சான் வேர்பட்டை	7½ வராகன்
தலைசுருளி வேர்பட்டை	7½ வராகன்
உலர்த்தின சங்கங்குப்பயிலை	7½ வராகன்
சங்கன் வேர்பட்டை	7½ வராகன்
உலர்த்தின வெள்ளருகு சமூலம்	7½ வராகன்
உலர்த்தின கையாந்தகரை சமூலம்	7½ வராகன்
செங்கத்தாரி வேர்பட்டை	7½ வராகன்

செய்முறை

மேற்கண்ட சரக்குகளை முறைப்படி சுத்தி செய்து சூரணித்துக் கொள்ள வேண்டும்.

அளவு

¼ - ½ வராகன் எடை

தீரும்பிணி

வெண்படை

Trial Medicine II (External)

Name of the Medicine: வெண்படைபற்று (வெண்குட்ட லேபனம்)

Reference : Patharthaguana vilakam Page No.234

சேரும் சரக்குகள்

காட்டு சீரகம் 40 கி

அரிதாரம் 10 கி

பசுநீர் தேவையான அளவு

செய்முறை

அரிதாரம் சுத்தி முறை

தாளகத்தை சன்னமாக வெட்டி இரட்டை மடிப்பு சேலையில் கட்டி - பசுநீர், அரிசி கழுவிய நீர், புளித்த காடி இவைகளில் ஒன்று மூன்று நாள் தோலாயந்திரமாக கமலாக்கனி கொண்டு எரித்து எடுக்க சுத்தியாகும்.

செய்முறை

மேற்கண்ட சரக்குகளை சுத்தி செய்து பசுவின் நீர்விட்டு குழம்பு பதமாக அரைத்து பற்று போட வேண்டும்.

அளவு தேவையான அளவு

தீரும் நோய் வெண்படை

2.REVIEW LITERATURE OF TRIAL DRUG

பறங்கிப்பட்டை

Bot Name	:	Smilax China
Family	:	Liliaceae
சுவை	:	இனிப்பு
தன்மை	:	தட்பம்
பிரிவு	:	இனிப்பு

செய்கை:

உடற்றேற்றி

மேகப்பிணிவிலக்கி

காமம் பெருக்கி

தூய்மையாக்கி

பொதுகுணம்:

“தாகம் பலவாதக் தாதுநட்டம் புண்பிளவை

மேகங் கடிகிரந்தி வீழ்மூலக் - தேகமுடன்

குட்டை பகந்தமேற் கொள்வமனம் போம் பறங்கிப்

பட்டையினை யுச்சரித்துப் பார்”

Constituents:

Root contains fat, sugar, glucoside, colouring matter, saponin (Sarasasapogenin) gum and starch. Including psoriasis, rheumatoid artaritis, gout, enteritis, urinary tract Infections, smilax china – skin ulcers.

The root is

- Alterative
- Anti scropulatic
- Carminative
- Depurative
- Diaphoretic
- Diunetic, Tonic

சங்கன் வேர்

Bot Name : Azima tetracantha

Family : Salvadoraceae

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை

சிறுநீர்ப்பெருக்கி

வெப்பமுண்டாக்கி

துவர்ப்பி

உரமாக்கி

முறைவெப்பமகற்றி

கோழையகற்றி

பொதுகுணம்:

“சங்கம் வேர்ப்படை சளியிருமலைச் சுரத்தை
அங்கவா தக்கடுப்பை ஆடதைப்பைப் பங்கமே
செய்யுங் கிரந்தியையுள் தீகால் கிருமியையிங்
வையந் தனிலொழிக்கு மால்” - அகத்தியர் குணவாகடம்

“வீக்கம் கரப்பான் விதாகம் கிரந்தி குன்மம்
ஊக்கமிகு சூலைவாய் வோடு பித்தத் - தாக்குவிடம்
வீறுமோ கண்துலங்கும் வீசபசி ரத்தமுண்டாம்
கூறுசங்கம் வேரிலை கட்கு – அகத்தியர் குணவாகடம்

Azima Tetra Cantha Linn:

High concentrations of N – methoxy – 3 – indolylmethyl – glucosinolate, a common glucosinate were found in the roots and seeds of a Tetra contha.

The seeds of Azima tetracantha contained a complex mixture of 267 lavonoids pre dominantly as glycosides.

Alkaloids :

Azimene, Zalarpine, carpine,

சிவனார்வேம்பு

Bot Name	:	Indigofera aspalathoides
Family	:	Fabaceae
சுவை	:	கைப்பு
தன்மை	:	வெப்பம்
பிரிவு	:	கார்ப்பு

செய்கை

வெப்பமுண்டாக்கி
வேர் - குளிர்ச்சியுண்டாக்கி
உள்ளலாற்றி

பொதுகுணம்

குட்டஞ் சிரங்கு குறைப்புப் பிசமாந்தை
கட்டப் பிணிகள் கழலுமே – திட்டம்
உரனிம்பங் காயத்துக் குண்டாகு மேலை
அரனிம்பு மென்னுமரு ந்தால் - தேரன் வெண்பா

சாய்க்குமிடிப் புண்பழம்புண் சர்மகுட் டம்பிளவை
தீக்கடுகால் வன்பெரு நோய் சிந்துமா நோய்க்கு
விதையாதி வேம்பினா மெய்க்கழகு காலை
உதையாதி வேம்பினா லுன்

- அகத்தியர் குணவாகடம்

- Leaves, flowers and tender shoots – coding, demulcent
- Employed in decotion in leprosy and cancerous affections.
- Indigofera aspalathoides works wonder in developing sustainable immunity in human body.
- Anti inflammatony agent
- Anti tuberulous agent
- Anti bacterial
- In activator of succinate dehydrogenase.

கரிசாலை

Bot Name : Eclipta prostrata

Family : Asteraceae (Compositate)

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை

பித்தநீர்ப் பெருக்கி
உரமாக்கி
உடற்றேற்றி
வாந்தியண்டாக்கி
நீர்மலம் போக்கி
வீக்கமுருக்கி
ஈரல்தேற்றி

பொதுகுணம்

குரற்கம்மல் காமாலை குட்டடிமாடு சோபை
யுறற்பாண்டு பன்னோ யொழிய - நிரற்சொன்ன
மெய்யாந் தகரையொத்த மீனி ண்ணு நற்புலத்துக்
கையாந் தகரையொத்தக் கால்

- அகத்தியர் குணவாகடம்

Constituents :

- A large amount of resin Alkarloid ecliptine
- Tonic and deobstruent
- Use in neputic and spleen encouragement
- Skin uleers
- Source of black stain
- Root emitic and purgative

வெள்ளறுகு

Bot Name	:	Enicostemma axillare
வேறு பெயர்	:	குலோமி, சண்டம், திட்டை, சிலேட்டு மந்தை, சக்கிர வீரியநந்தன்
சுவை	:	கைப்பு
தன்மை	:	வெப்பம்
பிரிவு	:	கார்ப்பு

செய்கை

பசித்தீத்தூண்டி

உரமாக்கி

உடற்றேற்றி

மலமிளக்கி

வெப்பமகற்றி

பொதுகுணம்

குன்மமொரு வாய்வு குடல்வாதம் சுவையிவை
சென்மம் விட் டோடிச் சிதையுங்காண் - வன்முலையாய்
உள்ளூறுகி ரந்திசொறி யொட்டிய சிரங்குமறும்
வெள்ளறுகு தன்னை விரும்பு

-குணபாடம் மூலிகை

Uses

- Dyspepsia, Flatulence
- Colic, Helminthiasis
- Abdominal ulcers, Hernia
- Snelling, alycosaria
- Leprosy, skin disease, pruritis
- Intermittent fever
- Local applications in snake poisons.

Phytochemicals

Ophelic acid, tanniens, gentiamine, erythodentawin, gentioorunine, apigenin

சங்கங்குப்பி

Bot Name	:	Clerodendrum inerme
Family	:	Verbenaceae
வேறு பெயர்	:	இசங்கு, முத்தாபலம், பீச்சங்கன், கோல்
சுவை	:	கைப்பு
தன்மை	:	வெப்பம்
பிரிவு	:	கார்ப்பு

செய்கை

உடற்றேற்றி

வெப்பகற்றி

உரமாக்கி (Tonic)

பொதுகுணம்

கரப்பான் கிரந்தி கருங்குட்ட ரோகம்
உரப்பான மேகம் ஒழியுங் கருவாம்
கருங்கிரந்தி செவ்வாப்புக் கட்டிகளு மேகும்
அருஞ்சங்கங் குப்பிக் கறி
வெட்டை சொறி சிரங்கு வீறி வருஞ்சூலை
துட்டவா தங்கபந்து ணுக்கிருமல் கெட்டவிடம்
அங்கங் கொள் பூச்சிவை யாவும்போம் பித்த முறுஞ்
சங்கங்குப்பிக் கெனவே சாற்று

Constituents:

Leaves contain a bitter principle

Resin

Gum

A brown colouring matter &

Ash containing a large amount of sodium chloride

This plant possesses therefore ecbolic, hypertensine, and laxative effects. The plant proved to be non – toxic since it does not produce ill effects with doses as loose as 8 g / kg body weight of the powdered plant.

செங்கத்தாரி

Bot Name : Capparis Sepiaria

Family : Capparideae

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : துவர்ப்பி உடற்றேற்றி

பொது குணம்

"செங் கத்தா ரிச்சடைக்குத் தீராத வன்மேகம்
பொங்கி வருங்கிரந்திப் புண் புரைகள் - தங்குகின்ற
சந்திகசி லேஷ்டிக் தனித்த மகாவத
முந்திவிட் டோடு மொழி

Constituents

Bark contains a neutral bitter principle resembling senegen

Flower buds : Contain capric acid & a glycoside which yield on boiling with sulphuric acid isodulite and a colouring matter similar to quercertin.

Alkaloids

A new spermidine alkaloid capparidisin isolated form root bark and its structure capprisinine.

B capparisine carotene isolated from leaves.

ஈச்சர மூலி

Bot Name	:	Aristolochia Indica Linn
Family	:	Aristolochiaceae
வேறு பெயர்	:	தலை சுருளி, பெருமருந்து, தராசுக்கொடி
சுவை	:	கைப்பு
தன்மை	:	வெப்பம்
பிரிவு	:	கார்ப்பு

செய்கை

வெப்பமுண்டாக்கி

உரமாக்கி

ருது உண்டாக்கி

பொதுகுணம்

பாண்டகற்று மெய்யிற் படர்குட்ட நோய் விலக்கும்
நீண்ட விருதய நோய் நீக்குங்காண் - தாண்டதப்பை
முன்னே யொழித்துவிடு மூவாத் தலைச் சுருளி
யென்னே யுலகி லிசை
பெருமருந்தின் வேர் பித்தம் பீறிரைப்பு காசம்
வருசுரம் உடம்பு வலி வாதம் உருவு விடம்
ஒன்றிய மாகழகள் ஓட்டும் உலகலிது
அன்றியது மீகாச் சிகிச்சைக்காம்.

Phytochemicals

- Arstolochin, tannin, isoaristolochic acid
- allantonin

Uses

- Root used in fever and leucodema
- Respriatory parlysis

சிறுகுறிஞ்சான்

Bot Name : Gymnema Sylvestre (ectz)

Family : Menispermeceae

Part used : Root

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை

வெப்பமுண்டாக்கி
துவர்ப்பி
வாந்தியுண்டாக்கி
பசித்தீதூண்டி
உரமாக்கி

பொது குணம்

வேர்

சிறு குளிஞ்சான் வேர் விடத்தைத் தீர்க்கும்
அனிலத் துறு சுரங்கன் வாதம் ஒழிக்குந் - தெளிபாணக்
கண்ணாய் இருமல் முதல் காச சுவாசந் தணிக்கும்
விண்ணா டருக்கிதனை விள்

- அகத்தியர் குணவாகடம்

Phytochemicals

Gymenmic acid, leaves contain anthroguione compound.

Uses

- Fever
- Anti helminthatic effect
- Cough
- Bronchial asthma
- Skin diseases

Venpadaipatru (External)

சேரும் சரக்குகள்

காட்டுச்சீரகம்

Bot Name	:	Verrnoia anthelmintica
Family	:	Asteraceae
சுவை	:	கைப்பு
தன்மை	:	வெப்பம்
பிரிவு	:	கார்ப்பு

செய்கை

புழுக்கொல்லி
பசித்தீத்தூண்டி
உரமாக்கி
சிறுநீர் பெருக்கி
முறைவெப்பமகற்றி
உடற்றேற்றி

பொதுகுணம்

கைகறுப்பு மாறுங் கடியமே கம்போகும்
மெய்குளிரும் பித்தம் விளையுமோ – வெய்யகரிக
கோட்டுப் பணைமுலையாய் குன்மவா தந்தொலையுங்
காட்டுநற் சீரகத்தைக் காண்

Constituents :

- Seeds contains resins, and alkaloid known as vernomine, an oil and ash amounting to about 7 P.C. of the dry material free from manganese.
- Avenasterol, renosterol, essential oil, resins, myristic, palmitic, stearic, oleic, lonoleic, vernolic acid.

தாளகம்

தாளகத்தின் வேறு பெயர்கள்

தமிழ் :

- மால்பாகம், மால்தேவி, அரிதாரம், இலட்சுமி, கோதந்தி, பீதகம், சரம், மிச்சிரம், கெந்தகம், நடமணடனம், பிடாலகம், சேல்கண்ணி, முக்காட்சி, ஆட்குடிச்சி, அரிசத்தி, கௌரவம்.
- பீதகி, ஆலம்பி, பிஞ்சனம், பழுப்பு, கோதந்தம், மாலம், அரிதாரம், கால்புத்தி, பொன்வர்ணி, மால்தேவி, அரிதளம்.
  மடந்தை, திருமடந்தை, நாரி, தேவி, விஷ்ணுவின், பெண்டிர், விஷ்ணுவின் மனைவி, வாழ்மடந்தை.

தாளகத்தின் பொதுக்குணம்:

தாளகத்தின் பேருரைக்கத் தாலுகவுள் நோய்குஷ்டம்
நீளக்குளிர்காய்ச்சல் நீடுகபம் - நாளகங்கொள்
துஷ்டபப் பறங்கிப்புண் சூழழுகண் மண்டைநோய்
கிட்டப் படுமா கிளத்து

தாளகத்தினால் நாக்கு, கபோலம் இவைகளைப் பற்றிய நோய், குட்டம், குளிர் சுரம், சுபம், மூத்திர நாளத்தைப் பற்றிய பறங்கிப் புண், அழுகண், மண்டைநோய் முதலியவை நீங்கும்

CHEMICAL ASPECT OF (ORPIMENT) THALAGAM

Orpiment Is A Rare Mineral.

Name	:	Orpiment
Synonyms	:	Arsenic trisulphide, Yellow arsenic sulfide, Natural arsenic suplphides, Arrhenicum, Operment, Orpiment, Yellow Arsenic
Name origin	:	The name is derived from the Latin " auripigmentium" ("aurum"-gold and pigmentum) relating to the minerals "goldish color". Orpiment is also known as "King's Yellow", "Chinese Yellow" and "Yellow Orpiment".
Colour	:	Lemon –yellow, Orange Orange-brownish, Deep orange-colored Orpiment crystal aggregate.

Chemical Formula:		A
Class	:	Sulfides and sulfosalts
Chemical	:	Arsenic Sulfode
Elements	:	As, S
Composition	:	Arsenic -60.90%(As)
		Sulfur -39.10%(S)
Common Minerals:		Hg, Ge, Sb
Associated Minerals:		Usually forms with realgar. In fact the two minerals are almost Always together. Other mineals are stivnite, pyrite, sphalerite, Calcite, antimony ores, barite and gypsum.
Hardness	:	1.5-2.0
Specific Gravity:		3.4-3.5
Density	:	3.49-3.56, Average =3.52
Melting point	:	300^{o}C
Boiling point		707^{o}C

பசுநீர் (cows urine)

சித்த மருந்துகளில் சுத்தி முறைகளுக்கு பயன்படுகிறது.

செய்கை

சிறுநீர் பெருக்கி

மலமிளக்கி

பொதுகுணம்

“விடபாண்டு சோபைபல வீக்கஞ் சகல
விடமுதிர மாலையென மெத்த-புடவிதநீர்
பேசலக்க ணோடுதந்தப் பீடை யகன்றிடுமே
கோசலத்த லாரணங்கே கூறு”

Chemical description of cow urine as per modern concepts and cure of diseases accordingly

Table - Chemical contents of cow urine and cure of diseases as per them

S. No.	Name of chemical	Effect of chemical on diseases
1.	Nitrogen N2 ,NH2	Removes blood abnormalities and toxins, Natural stimulant of urinary track, activates kidneys and it is diuretic.
2.	Sulphur S	Supports motion in large intestines. Cleanses blood.
3.	Copper Cu	Controls built up of unwanted fats
4.	Iron Fe	Maintains balance and helps in production of red blood cells & haemoglobin. Stabilises working power.
5.	Urea CO(NH2)2	Affects urine formation and removal. Germicidal.
6.	Potassium K	Cures hereditary rheumatism. Increases appetite. Removes muscular weakness and laziness.
7.	Calcium Ca	Blood purifier, bone strengthener, germicidal
8.	Salt NaCl	Decreases acidic contents of blood, germicidal
9.	Vitamins A,B,C,D,E	Vitamin B is active ingredient for energetic life and saves from nervousness and thirst, strengthens bones and reproductive ingredient for energetic life and saves from nervousness and thirst, strengthens bones and reproductive power.
10.	Enzymes	Make healthy digestive juices, increase immunity

The invention relates to an absolutely novel use of cow urine distillate as activity enhancer and availability facilitator for bioactive molecules including anti-infective and anti-cancer agents. The molecules which express any activity in form of either inhibiting or promoting a biological function have been referred in this invention as bioactive molecule e.g. antibiotics, drugs, nutraceuticals, cardiovascular, hepatoprotective, neuro-tonics etc. The present invention has direct implication in drastically reducing the dosage of antibiotics, drugs and anti-cancer agent while increasing the efficiency of absorption of bioactive molecules.

3.ANTIMICROBIAL ACTIVITY OF VENPADAI PATTRU

Venkutta Lapanam

The anti microbial activity of different extracts were studied by disc diffusion method against the following organisms.

Media used	–	Nutrient Agar.
Ingredients		gms/litre.
Peptone		10.00
Beet extract		10.00
Sodium chloride		5.00
Agar		12.00

Disc diffusion method :

A suspension of staphylococcus aureus was added to sterile nutrient agar at 45’c the mixture was transferred to sterile petridishes and allowed to sterile discs 5mm in diameter (made from what man filter paper previously sterilized in u-v- lamp dipped in solution of the different extracts standard on the surface of agar plates .

Leave the plates were incubated at 37’c for 24 hours and observed for antibacterial activity the diameters of the zones of in inhibition Was measured for the plates in which the zone of inhibition was observed .

The average area of zone of inhibition was calculated and compared with that of the standard

A similar procedure was carried out for the study of antibacterial activity of this sampler against mentioned organisms.

Antimicrobial Activity OfVenpadai Pattru

S. No	Organisms gram +ve.	Std	50	100	150
1	Staphylococcs aureus	32	16	21	26
2	Staphylococcus epidermidis	31	15	19	25
3	Bacillus cereus	33	18	21	28
4.	Bacillus subtilis	32	17	22	21
5.	Micrococcus luteus	34	20	24	28
6.	Streptococcus mutans	32	21	25	28

(Gram –ve)

1	Escherichia coli	33	15	19	24
2.	Klebsiella pneumonial	32	15	18	23
3.	Pseulomonas aeruginosa	32	14	18	22

Bacterial:

Standard used:

Ciproflaxacin hcl 50 mcg/disc.

4. ANTIFUNGAL ACTIVITY OF VENPADAI PATTRU

(VENKUTTAL LAPANAM)

The anti fungal activity of different extracts were studied by disc diffusion method against the following organisms.

Media used :

Sabouraud dextrose agar medium gm/ litre.

Mycological peptone	10.0
Dextrose	40.0
Agar	15.0

DISC DIFFUSION METHOD :

A suspension of A. Niger was added to sterile sabouraud dextrose agar at 45. the mixture was transferred to sterile petridishes and allowed to sterile petridishes and allowed to solidity, sterile disc 5mm in diameter (made from whatman fitter paper previously sterilized in u-v- lamp) dipped in solution of the different concentrations, standards and a blank were placed on the surface of agar plates

Leave the plates standing for 4 hours at room pre-incubation diffusion o minimize the effects of variations in time between the application of the different solutions, then the plates were incubated at 37'c for antibacterial activity

the diameter of the zones of inhibition was observed .

The average area of zone of inhibition was calculated and compared with that of the standards.

A similar procedure was carried out for the study of anti fungal of extract against aspergillus fumigates & candida albicans

ANTIFUNGAL ACTIVITY OF VENPADAI PATTRU

(VENKUTALAPANAM)

Fungus

1.	Aspergillus niger	29	16	18	22
2.	Aspergillus fumigates	29	17	19	23
3.	Candida albicans	33	18	22	25

Fungus :

Standard used:

Ketoconazole → 50 mcg/disc

Sample concenatration

1 gm → 100ml solvent water 50,100,150 ul/disc

5.BIO CHEMICAL ANALYSIS OF LAGU SEENA CHOORANAM

Physical properties:

Loss on drying:

Five grams of Lagu seena chooranam is heated in a hot oven at 40^{o}C to constant weight. The percentage of loss of weight was calculated.

Determination of ash value:

Weigh accurately 2-3 grams of Lagu seena chooranam in tarred platinum or silica dish and incinerate at a temperature not exceeding 450^{o}C until free from carbon, cool and weigh. Calculate the percentage of ash with reference to the air dried drug.

Acid Insoluble ash

Boil the ash for 5 minutes with 25 ml of 1: 1 dilute HCL. Collected the insoluble matter in Gooch – crucible on an ash less filter paper, wash with hot water and ignite, cool in a dessicator and weigh. Calculate the percentage of acid insoluble ash with reference to the air dried drug.

Water soluble ash:

To the Gooch crucible containing the total ash, add 25 ml of water and boil for 5 minutes. Collect the insoluble matter in a sintered glass crucible or on ash less filter paper. Wash with hot water and ignite in a crucible for 15 minutes at a temperature not exceeding 450^{o}C. Subtract the weight of the insoluble matter from the weight of the ash; the difference of weight represents the water soluble ash. Calculate the percentage of water soluble ash with reference to the air dried drug.

Alkalinity of water soluble ash:

Five grams of Lagu seena chooranam converted to ash, boiled with water, filtered. Filtrate was titrated against 0.1N of HCl using phenolphthalein as an indicator.

Alkalinity of water soluble ash = X x of acid / 0.1 x W

X = Titre value.

W = Weight of the material taken.

Alkalinity is given as ml of 0.1N of HCL equated to 1 gm.

PH:

Five grams of Lagu seena chooranam is weighed accurately and placed in clear 100 ml beaker. Then 50 ml of distilled water is added to it and dissolved well. Wait for 30 minutes and then apply in to pH meter at standard buffer solution of 4.0, 7.0, and 9.2.

QUANTITATIVE ANALYSIS

Sl. No.	**PARAMETER**	RESULTS (%)
01.	Loss of drying @ 105^{o}C	6.72
02.	Ash Value	12.89
03.	Water soluble	9.80
04.	Alkalinity as CaCO3 in water soluble ash	0.05
05.	Acid insoluble ash	5.25
06.	PH at 10% aqueous solution	5.68

QUALITATIVE ANALYSIS

SL.NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	Appearance of the sample	Brown in colour	
2.	Solubility: a. A little of the sample is shaken well with distilled water. b. A little of the sample is Shaken well with con. Hcl Con. H_2SO_4.	 Completely soluble Completely soluble	 Absence of Silicate
3.	Action of Heat: A small amount of the sample is taken in a dry test tube and heated gartly at first and then Strong.	 White fumes not evolved Brown fumes evolved	 Absence of Carbonate. Presence of Nitrate.
4.	Flame Test: A small amount of the sample is made into a paste with con. Hcl in a watch glass and introduced into non-luminous part of the Bunsen flame.	 White flame is appeared	 Absence of Copper.
5	Ash Test: A filter paper is soaked into a mixture of sample and cobalt nitrate solution and introduced into the Bunsen flame and ignited	No Yellow colour flame.	Absence of Sodium.

Preparation of the extract

5 gm of Lagu seena chooranam was weighed accurately and placed in a 250 ml clean beaker. Then 50 ml distilled water was added and dissolved well. Then it is boiled well for about 10 minutes. It was cooled and filtered in a 100 ml volumetric flask and then it was made up to 100 ml with distilled water. This fluid was taken for analysis.

SL.NO	EXPERIMENT	OBSERVATION	INFERENCE
TEST FOR ACID RADICALS			
1.	**Test For Sulphate:** a. 2 ml of the above prepared extract is taken in a test tube to this added 2ml of 4% ammonium oxalate solution b. 2 ml of the above prepared extract is added with 2 ml of dil-Hcl is added until the effervescence ceases off. Then 2ml of Barium chloride solution is added	Cloudy appearance present A white precipitate insoluble in con. Hcl is obtained	**Presence of Sulphate**. Sulphate is confirmed.
2.	**Test For Chloride:** 2 ml of the above prepared extract is added with dil. HNO_3 till the effervescence ceases. Then 2 ml of silver nitrate solution is added.	No Cloudy appearance present	Absence of Chloride.

3.	**Test For Phosphate:** 2 ml of the extract is treated with 2ml of ammonium molybdate solution and 2 ml of con. HNO_3	No cloudy yellow appearance	Absence of Phospate.
4.	**Test For Carbonate:** 2m1 of the extract is treated with 2m1 magnesium sulphate solution	No cloudy appearance	Absence of Carbonate.
5	**Test For Nitrate:** 1gm of the substance is heated with copper turnings and concentrated H_2SO_4 and viewed the test tube vertically down.	Brown gas is evolved	**Presence of Nitrate.**
6.	**Test For Sulphide:** 1 gm of the substance is treated with 2m1 of con. Hcl.	No Rotten egg smelling gas evolved	Absence of Sulphide.
7.	**Test For Fluoride & Oxalate** 2 m1 of The Extract Is Added With 2m1of Acetic Acid and 2 m1 calcium Chloride solution and heated.	No Cloudy appearance.	Absence of Fluoride & Oxalate
8.	**Test For Nitrite** 3drops of extract is placed on a filter paper, n that 2 drops of acetic Acid and 2 drops of benzidine solution is placed.	No characteristic changes	Absence of nitrite.

9.	**Test For borate:** 2 pinches of the substance is made into paste by using sulphuric acid and alcohol (95%) and introduced into the blue flame.	Bluish green colour flame not appeared..	Absence of borate.
II. TEST FOR BASIC RADICALS			
1	**Test For Lead:** 2 m1of the extract is added with 2m1of potassium iodide solution.	No Yellow precipitate is obtained	Absence of Lead.
2.	**Test for Copper:** a. one pinch of substance is made into paste with con. Hcl in a watch glass and introduced into the non-luminous part of the flame. b. 2 ml of extract is added with excess of ammonia solution.	No Blue colour flame precipitate No Blue colour precipitate	Absence of Copper. Absence of Copper.
3.	**Test For Aluminium:** Take the 2m1of the extract sodium hydroxide is added in drops to excess.	No characteristic changes	Absence of Aluminium.
4.	**Test For Iron: (Ferrous)** To the 2 ml of extract 2m1 ammonium thiocyanate solution and 2m1of con.HNO_3 is added	Blood red colour Appearance	**Presence of Iron.**

5.	**Test For Zinc:** To 2m1 of the extract sodium hydroxide solution is added in drops to excess.	White precipitate is not Formed	Absence of Zinc.
6.	**Test For Calcium:** 2m1 of the extract is added with 2m1 of 4% ammonium oxalate Solution.	Cloudy appearance and white precipitate is obtained	Presence of Calcium.
7.	**Test For Magnesium:** To 2ml of extract sodium hydroxide solution is added in drops to excess.	White precipitate is not obtained.	Absence of Magnesium.
8.	**Test For Ammonium:** To 2ml of extract few ml of Nessler's reagent and excess of sodium hydroxide solution are added.	No Brown colour appeared.	Absence of Ammonium.
9.	**'Test For Potassium:** A pinch of substance is treated with 2ml of sodium nitrite solution and then treated with 2m1 of cobalt nitrate in 30% glacial acetic acid.	No Yellowish precipitate is obtained	Absence of Potassium.

10.	**Test For Sodium:** 2 pinches of the substance is made into paste by using HCL and introduced into the blue flame of Bunsen burner.	No Yellow colour Flame appeared.	Absence of Sodium.
11.	**Test For Mercury:** 2m1 of the extract is treated with 2ml of sodium hydroxide solution.	No Yellow precipitate is obtained	Absence of Mercury.
12.	**Test For Arsenic:** 2m1 of the extract is treated with 2m1 of sodium hydroxide solution.	No brownish red Precipitate is obtained	Absence of Arsenic.
III. MISCELLANEOUS			
1.	**Test for Starch:** 2ml of extract is treated with weak iodine solution.	No blue colour developed	Absence of Strarch.
2.	**Test For Reducing Sugar:** 5. ml of Benedict's qualitative solution is taken in a test tube and	No colour Changes	Absence of Reducing sugar.

	allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boil it for 2 minutes. The colour changes are noted.		
3.	**Test For The Alkaloids:** a. 2m1 of the extract is treated with 2ml of potassium Iodide solution. b. 2m1 of extract is treated with 2ml of picric acid. c. 2m1 of the extract is treated with 2ml of phosphotungstic acid.	No Red colour developed Trace Yellow colour developed No white precipitate developed	Absence of Alkaloid. Trace of Alkaloid present. Absence of Alkaloid
4.	**Test for Tannic Acid:** 2ml of extract is treated with 2ml of ferric chloride solution.	No black precipitate is obtained	Absence of Tannic acid.
5.	**Test for Unsaturated Compound:** To the 2ml of extract 2ml of Potassium Permanganate solution is added.	Potassium Permanganate is not decolourised	Absence of Unsaturated Compound.

6.	**Test For Amino Acid:** 2 drops of the extract is placed on a filter paper and dried well and 2 ml of biuret reagent is added.	No Violet colour developed	Absence of Amino acids.
7.	**Test For type of Compound:** 2ml of the extract is treated with 2 ml of ferric chloride solution.	No Green colour developed No Red colour developed No Violet colour developed No blue colour developed	Absence of oxy quinole epinephrine and pyro catechol. Anti pyrine, Aliphatic amino acids and Meconic acid are absent. Apomorphine, Salicylate and Resorcinol are absent. Morphine, Phenol cresol and hydro quinone are absent

RESULT:

Iron, Calcium, Sulphate, Nitrate, Traces of Alkaloids, Compounds are present in **lagu seena chooranam.**

6.TOXICOLOGICL EVALUATION

ACUTE ORAL TOXICITY STUDY OF LAGU SEENA CHOORANAM

Experimental Animals

Adult female wistar albino rats (150-230 gm) were obtained from the animal housing facility of King Institute, Guindy, Chennai. The animals were maintained in a well – ventilated room with 12:12 hour light/dark cycle in polypropylene cages. Standard pellet feed and tap water were provided *ad libitum* through out experimentation period. Animals were acclimatized to laboratory conditions one week prior to initiation of experiments. The project has got the ethical committee clearance from IAEC (Institutional Animal Ethics Committee) of CPCSEA (Committee for the Purpose of Control and Supervision of Experiments on Animals).

Acute oral toxicity study (Ecobichnon, 1997)

The procedure was followed by using OECD guidelines (Organization of Economic Cooperation Development)-423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 animals of a single sex per step. Depending on the mortality and/or moribund status of the animals, on the average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance . This procedure results in use of minimal number of animals while allowing for acceptable data based scientific conclusion. The method used defined doses (5, 50, 300, 2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which cause acute toxicity.

Preparation of Drug for administration to animals:

Lagu seena chooranam suspended in 1% sodium carboxyl methyl cellulose solution was used for the experimental studies.

Experimental Procedure:

Female wistar rats weighing about 150-200 g were used for the study. The starting dose level used was 2000 mg/kg p.o. Dose volume was administered 0.1 ml/ 10 gm body weight to the rat which were fasted over night with water *ad libitum.* Food was withheld for a further 3-4 hours after administration of ***Lagu seena chooranam*** and observed for signs of toxicity. Body weight of the rats before and after termination were noted and any changes in skin and fur, eyes and mucous membranes and also respiratory, circulatory, autonomic and central nervous system and somatomotor activity and behavior pattern were observed, and also sign of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma were noted. The onset of toxicity and signs of toxicity also noted.

RESULT

The Acute oral toxicity study was done according to the OECD guidelines 423(Acute toxic classic method) A starting dose of 2000 mg /kg b.w.p.o of *Lagu seena chooranam* was administered to 3 female rats observed for 14 days .There was no considerable change in body weight before and after treatment of the experiment and no signs of toxicity were observed .When the experiments were repeated again with the same dose level 2000 mg/kg b.w.p.o of Lagu seena chooranam observed for 14 days ,no changes were observed.so the LD_{50} cut off value mg /kg body weight was observed as **X**-un classified

Ref: Ecobichon DJ. The basis of Toxicity Testing 2nd editors New york. CRC press 1998. Pg.no. 43-88.

Out patient Department

Before Treatment

After Treatment

S.NO	OPNO	Age/ Sex	TC	DC			ESR		HB	RBC	TC	DC			Esr		Hb	Rbc	Before Ofter			After Before		
				P	L	E	½	½				P	L	E	½	½			Alb	Sug	Dep	Alb	Sug	Dep
1.	AM1932	8/F	6100	54	44	2	4	8	11.1	3.3	6700	57	40	3	10	22	11.0	2.4	Nil	Nil	2-3 2-3	Nil	Nil	1-2 2-4
2	AM1872	12/M	6000	51	47	2	4	8	9.1	3.2	6500	55	43	3	4	8	10.5	3.5	Nil	Nil	1-2 2-4	Nil	Nil	1-2 1-2
3	AM1978	12/F	7500	58	40	2	10	20	9.7	3.2	7300	53	46	1	3	6	10.5	3.6	Nil	Nil	2-4 1-2	Nil	Nil	1-2 1-2
4	AM2647	10/F	7000	53	43	4	2	4	12.6	4.2	7000	51	47	2	4	10	12.5	3.4	Nil	Nil	1-2 1-2	Nil	Nil	2-3 1-2
5	AM2528	9/M	7400	52	47	1	4	10	10.7	3.6	7700	50	48	2	4	10	14	4.8	Nil	Nil	3-5 2-3	Nil	Nil	1-3 0-1
6	AM2603	8/F	6000	54	44	2	10	20	9.7	3.2	6300	54	44	2	4	8	10.5	3.6	Nil	Nil	2-4 2-4	Nil	Nil	1-2 0-1
7	AM2523	10/M	7200	54	42	4	4	8	10.2	3.2	7100	52	44	4	6	12	10.5	3.4	Nil	Nil	6-8 2-3	Nil	Nil	1-2 0-1
8	AM2579	7/F	6100	50	45	5	4	8	11.6	3.6	7800	50	49	1	4	8	10.9	3.2	Nil	Nil	2-3 2-3	Nil	Nil	2-3 1-5
9	AM2521	12/M	6200	60	35	5	4	10	12.1	4.0	7600	50	46	4	6	12	12.2	3.4	Nil	Nil	2-3 2-3	Nil	Nil	1-2 1-2
10	AM2634	10/M	6000	51	44	5	4	8	10.2	3.1	7100	51	47	2	4	8	10.5	3.4	Nil	Nil	4-5 2-3	Nil	Nil	1-2 1-2
11	AM2606	6/F	7000	50	40	10	10	20	11.6	3.8	6200	51	46	3	4	8	10.2	3.4	Nil	Nil	2-3 2-3	Nil	Nil	1-2 1-2
12	AM3302	12/F	6100	50	45	5	4	8	9.2	3.0	7100	50	47	3	4	8	14.0	4.5	Nil	Nil	3-4 2-3	Nil	Nil	1-2 1-2
13	AM4669	8/F	8000	60	38	2	20	40	9.7	3.1	7500	58	40	2	6	12	10.5	3.5	Nil	Nil	1-2 1-2	Nil	Nil	1-2 0-7
14	AM4615	8/M	8500	60	39	1	15	32	12.6	4.1	8300	52	47	1	4	8	10.5	3.1	Nil	Nil	1-2 1-2	Nil	Nil	1-3 1-2
15	AM4859	12/M	7100	55	42	3	6	12	9.7	3.2	7400	54	44	2	8	16	14.0	4.5	Nil	Nil	1-2 2-4	Nil	Nil	1-2 2-4
16	AM4872	6/F	6100	58	40	2	2	4	8.7	2.9	7000	53	44	3	4	8	12.5	4.1	Nil	Nil	1-2 2-4	Nil	Nil	2-3 2-3
17	AM5886	11/F	6900	58	39	3	6	12	10.6	3.5	6800	56	43	1	4	8	11.0	4.0	Nil	Nil	1-2 1-2	Nil	Nil	1-2 2-4
18	AM4822	11/M	7900	62	35	3	6	12	10.6	3.4	7600	60	38	2	6	12	10.8	3.6	Nil	Nil	1-2 1-2	Nil	Nil	2-4 1-2

Out patient Department			Before Treatment								After Treatment													
19	AM8508	10/F	6000	57	39	4	4	8	10.6	3.5	6800	58	40	2	4	8	10.8	3.6	Nil	Nil	2-3 1-2	Nil	Nil	1-2 1-2
20	AM9882	9/M	7400	56	42	2	2	4	12.1	4.0	7200	58	40	2	6	12	11.5	3.8	Nil	Nil	1-3 0-1	Nil	Nil	3-5 2-3
21	AM70	11/F	7000	50	46	4	6	12	10.2	3.4	7900	54	44	2	6	12	13.0	4.3	Nil	Nil	1-2 0-1	Nil	Nil	2-4 2-4
22	AN47	7/F	6700	56	40	4	16	32	10.6	3.5	6200	50	45	3	8	20	10.6	3.5	Nil	Nil	1-2 0-1	Nil	Nil	6-8 2-3
23	AM9954	6/M	7100	52	45	3	4	8	10.6	3.5	8000	56	42	8	8	14	10.8	3.3	Nil	Nil	2-3 1-5	Nil	Nil	2-3 2-3
24	AN811	8/M	6700	50	45	5	10	20	9.7	3.2	6800	54	44	2	4	8	10.5	3.6	Nil	Nil	1-2 1-2	Nil	Nil	2-3 2-3
25	AN1025	9/F	6100	51	43	6	6	12	9.2	3.0	6500	55	43	2	6	2	10.2	3.2	Nil	Nil	1-2 1-2	Nil	Nil	4-5 2-3
26	AN1183	8/F	7100	58	40	2	2	4	10.6	3.4	6000	50	47	3	4	8	11.6	3.7	Nil	Nil	1-2 1-2	Nil	Nil	2-3 2-3
27	AN3767	12/M	7600	57	40	3	4	8	11.8	3.9	7000	58	40	2	4	10	11.5	2.7	Nil	Nil	1-2 1-2	Nil	Nil	3-4 2-3
28	AN1278	7/F	6200	56	42	2	2	4	9.2	3.0	7100	56	39	5	2	4	13.0	4.0	Nil	Nil	1-2 0-7	Nil	Nil	1-2 1-2
29	AN2416	8/M	6200	59	40	1	12	24	9.7	3.2	6800	60	38	2	4	8	10.5	3.6	Nil	Nil	1-3 1-2	Nil	Nil	1-2 1-2
30	AN2811	8/F	7300	57	41	2	2	4	10.6	3.5	7400	56	40	4	2	4	11.0	3.8	Nil	Nil	1-2 1-2	Nil	Nil	1-2 2-1

IN PATIENT DEPARTMENT

Before treatment **After Treatment**

S. NO	IP NO	Age/ Sex	TC	DC			ESR		HB	RBC	TC	DC			Esr		Hb	Rbc	Before Ofter			After Before		
				P	L	E	½	½				P	L	E	½	½			Alb	Sug	Dep	Alb	Sug	Dep
1.	241	9/F	7200	52	44	4	14	28	9.2	3.0	7,300	55	43	2	8	16	10.5	3.6	Nil	Nil	0-2 0.1	Nil	Nil	2-0
2.	243	12/F	6900	55	43	2	8	16	11.6	3.8	7,000	54	44	2	4	8	11.5	3.6	Nil	Nil	1-2 0-1	Nil	Nil	2-1
3.	247	9/M	6700	62	35	3	6	12	9.7	3.2	6.,900	60	38	2	3	6	12.1	3.5	Nil	Nil	1-2 0.1	Nil	Nil	1-2
4.	249	6/M	6700	52	46	2	18	36	8.7	2.5	6.,900	60	35	5	2	4	10.0	3.0	Nil	Nil	2-3 1-2	Nil	Nil	1-0
5.	253	12/M	6000	46	49	5	16	32	8.7	2.7	6,500	55	35	5	2	8	10.5	3.2	Nil	Nil	1-2 0-.1	Nil	Nil	1-2
6.	246	6/M	7000	56	40	4	10	20	8.2	3.2	7,200	58	38	4	4	4	10.6	3.2	Nil	Nil	Nil	Nil	Nil	
7.	262	11/F	7800	53	42	5	8	16	12.5	4.0	7,500	54	41	4	2	12	12.0	3.6	Nil	Nil	2-3 1-2	Nil	Nil	2-0
8.	255	12/M	6500	52	43	5	4	8	10.6	4.0	6,800	55	45	5	6	4	10.8	4.0	Nil	Nil	1-3 1-2	Nil	Nil	1-2
9.	245	10/F	6600	54	43	3	8	10	8.2	3.4	6,700	56	42	2	2	8	10.9	3.8	Nil	Nil	4-6 2-4	Nil	Nil	1-0
10.	266	6/F	8000	53	42	5	8	16	10.3	3.8	7.500	53	42	5	4	8	11.0	4.0	Nil	Nil	1-2 2-4	Nil	Nil	1-2
11.	269	8/F	8000	52	40	8	6	12	10.4	3.5	8,000	52	40	8	4	8	11.5	4.0	Nil	Nil	0-1 1-2	Nil	Nil	0-2
12.	272	6/F	7800	54	41	4	10	20	12.0	4.0	7,500	53	43	4	4	8	12.0	4.0	Nil	Nil	0-1 1-2	Nil	Nil	2-0
13.	274	9/M	7400	53	42	5	6	12	11.5	3.4	7,200	52	46	2	4	12	11.5	3.5	Nil	Nil	0-1 1-2	Nil	Nil	1-2
14.	276	11/F	6800	53	41	6	14	28	9.5	3.0	7,000	55	43	2	6	10	11.0	3.5	Nil	Nil	1-2 1-2	Nil	Nil	1-0
15.	285	10F	5200	59	37	4	30	60	11.2	4.0	6,000	55	44	1	5	8	11.5	4.0	Nil	Nil	1-2 1-2	Nil	Nil	1-2
16.	282	12F	6000	55	44	1	20	40	10.8	3.5	6,200	53	43	4	4	20	11.0	3.8	Nil	Nil	2-4 2-4	Nil	Nil	2-0
17.	295	8/M	7000	52	46	2	4	8	10.2	3.2	7,200	54	44	2	10	4	11.5	3.5	Nil	Nil	4-6 2-4	Nil	Nil	1-2
18.	326	8/M	5400	55	45	5	11	22	11.0	3.0	6,500	55	43	2	2	8	11.5	3.0	Nil	Nil	2-4	Nil	Nil	1-0
19.	330	7/F	6000	58	40	2	4	8	11.0	3.5	6,200	53	43	4	4	8	11.0	3.5	Nil	Nil	1-2 2-4	Nil	Nil	1-0
20.	332	6/M	6500	60	38	2	4	8	10.0	4.0	6300	58	38	4	4	8	10.5	3.8	Nil	Nil	1-2 2-4	Nil	Nil	0-2

7.STATISTICAL ANALYSIS

Paired t test – comparison of HB before and after treatment.

S .NO	TREATMENT	MEAN+ STD	T	P
1	Before	10.40+ 1.13	-4.58	<0.0001
2.	After	11.33+ 1.01		

The H B before and After Treatment is statistically highly significantPaired - t - test Treatment was used to determine the significant different.

A PILOT OPEN CLINICAL TRIAL OF *LAGU SEENA CHOORANAM* AND *VENPANDAI PATTRU (Venkuttalapanam)* FOR THE TREATMENT OF *VENADAI (*VITILIGO)

FORM I-SELECTION PROFORMA

1. OP/IP No:_____ 2.BED No:_____3.S.No:_____

4. NAME:_____5.AGE:_____Yrs 6.GENER:_____

7. DATE OF ADMISSION TO THE TRIAL | | | | | | |
Parent's/ Guardian's/ Name

8.PARENT'S OCCUPATION _____

9. INFORMATION ADDRESS: _____

INFORMATION RELATIONSHIP _____

COMPLAINS & DURATION: _____

……
……
……
……

11. HISTORY OF PRESENT ILLNESS:

……
……
……
……
……

12. PAST HISTORY:

……
……

13. FAMILY HISTORY:

…….
……

14. SOCIAL HISTORY:

Low socio economic (1) ☐ Middle class (2) ☐ Higher class (3) ☐

IMMUNISATION HISTORY Yes(1) ☐ No(2) ☐

HABITS: Yes(1) No(2)

15. Non-Vegetarian ☐ ☐

II) GENERAL EXAMINATION:

16. Body weight (kg) : ☐☐

17. Body height (cm) : ☐☐☐

17. Body temperature (F) : ☐☐☐☐

18. Blood Pressure (mmHg) : ☐☐☐ / ☐☐☐

19. Pulse Rate/min : ☐☐☐

20. Heart Rate/min : ☐☐☐

21. Respiratory Rate/min : ☐☐

		Yes (1)	No(2)
22. Pallor	:	☐	☐
23 Jaundice	:	☐	☐
24 . Clubbing	:	☐	☐
25. Cyanosis	:	☐	☐
26. Pedal Oedema	:	☐	☐
27. Lymphadenopathy	:	☐	☐
28. Jugular venous pulsesation	:	☐	☐

III) CLINICAL EXAMINATION OF SKIN:

29. ANATOMICAL LOCATION…….

30. COLOUR -Normal ☐ Hyperpigmented ☐ Hypopigmented ☐

31. SIZE OF THE LESION (Length cm): ……

32. SHAPE: Irregular ☐ Round ☐ Dispersed ☐

33. PRURITUS: Present ☐ Absent ☐

34. SWELLING: Present ☐ Absent ☐

35. ERYTHEMA: Present ☐ Absent ☐

36. DEPIGMENTATION OF HAIR

Present ☐ Absent ☐

37. SENSATION

Normal ☐ Paraesthesia ☐ Numbness ☐

Painful ☐ Burning ☐ Pricking ☐

38. SCALING: Present ☐ Absent ☐

39. CRUSTING: Present ☐ Absent ☐

40. OOZING: Present ☐ Absent ☐

41. MACULES: Present ☐ Absent ☐

42. PAPULES: Present ☐ Absent ☐

43. VESICLES: Present ☐ Absent ☐

44. PUSTULES: Present ☐ Absent ☐

45. PALPATION:

Normal ☐ Smooth ☐ Rough ☐

Warm ☐ Cold ☐

IV) EXAMINATION OF VITAL ORGANS:

	Normal (1)	Abnormal (2)	
46. CNS	☐	☐	...……
47. CVS	☐	☐	……..
48. RS	☐	☐	…….....
49. ABDOMEN	☐	☐	……..

SIDDHA ASPECTS

50. NILAM:

1.Kurinji ☐ 2.Mullai ☐ 3.Marutham ☐ 4.Neithal ☐ 5.Palai ☐

51. KALA IYALBU:

1.Karkaalam ☐ 2.Koothirkaalam ☐ 3.Munpanikaalam ☐

4.Pinpanikaalam ☐ 5.Illavenilkaalam ☐ 6.Muthuvenilkaalam ☐

52. UDAL IYALBU:

1.Vadam☐ 2.Pitham ☐ 3.Kabam ☐ 4.Vathapitham ☐ 5.Vathakabam ☐

6.Pithavadam ☐ 7.Pithakabam ☐ 8.kabavadham ☐ 9.Kabhapitham ☐

53.GUNAM:

1. Sathuvam☐ 2.Rasatham ☐ 3.Thamasam☐

AYMPORIGAL:

	Normal(1)	Affected(2)	
54. Mei	☐	☐	.....
55. Vaai	☐	☐	……
56 Kan	☐	☐	……
57. Mookku	☐	☐	……
58. Sevi	☐	☐	…….

VI) KANMENTHIRIUM / KANMAVEDAYAM

	Normal(1)	affected(2)	
59. Kai	☐	☐	……..
60 Kaal	☐	☐	……..
61. Vaai	☐	☐	……..
62. Earuvai	☐	☐	……..
63 Karuvai	☐	☐	……..

UYIR THATHUKKAL:

VATHAM:

	Normal (1)	affected (2)	
64 Pranan	☐	☐	…….
65 Abanan	☐	☐	……..
66.Viyanan	☐	☐	……..
67.Uthanan	☐	☐	……..
68.Samanan	☐	☐	……...

69.Nagan ☐ ☐ ……..

70.Koorman ☐ ☐ ……..

71.Kirukaran ☐ ☐ ……

72.Devathathan ☐ ☐ …….

73.Dhananjeyan ☐ ☐ ……..

PITTHAM:

Normal (1) affected (2)

74.Anar pittham ☐ ☐ ……..

75.Ranjagam ☐ ☐ ……

76.Sathagam ☐ ☐ …….

77.Alosagam ☐ ☐ ……

78.Prasagam ☐ ☐ …….

KABAM:

Normal (1) affected (2)

79.Avalambagam ☐ ☐ ……..

80.Kilethagam ☐ ☐ …….

81.Pothagam ☐ ☐ ……

82.Tharpagam ☐ ☐ …….

83.Santhegam ☐ ☐ ……

UDAL THATHUKKAL:

Normal (1) affected (2)

84. Saaram ☐ ☐ ……

85.Senneer ☐ ☐ …….

86.Oon ☐ ☐ …….

87.Kozhuppu ☐ ☐…….

88.Eanbu ☐ ☐ ……

89..Moolai ☐ ☐ ……

90.Sukkilam/ ☐ ☐

Suronitham ……..

ENVAGAI THERVUGAL:

91. Naadi …….

	Normal (1)	affected (2)
92.Naa	☐	☐
93. Niram	☐	☐
94.Mozhi	☐	☐
95.Vizhi	☐	☐
96.Sparisam	☐	☐

Malam:

	Normal (1)	affected (2)
97. Niram	☐	☐

	Yes(1)	No(2)
98. Nurai	☐	☐
99. Kirumi	☐	☐
100. Kalappu	☐	☐
101. Erugal	☐	☐
102. Elagal	☐	☐

Mootheram:

Neerkuri:

	Normal (1)	affected (2)
103. Niram =	☐	☐
104.Manam -	☐	☐
105.Edai	☐	☐
106.Nurai	☐	☐
107.Enjal	☐	☐

Neikuri: Vatha neer(1)☐ Pitha neer(2)☐ Kaba neer(3)☐

LAB INVESTIGATION:

BLOOD:

108. TC (Cells/Cu mm) ☐☐☐☐

DC (%)- 113.N-☐☐ 114.L-☐☐ 115..M-☐☐ 116.E -☐☐ 117.B-☐☐

☐☐ ☐☐

ESR (mm)- 118. 1/2 hr- 119.1 hr-

109. Hb (gm%)-

RBC (Millieum / Cubic)

URINE: Present (1) Absent (2)

110.Albumin ☐ ☐ …….....

111.Sugar ☐ ☐ ……

Deposit Present (1) Absent (2)

112.Pus cells - ☐ ☐ ……

113.Epithelial cells - ☐ ☐ ……

114.Red cells - ☐ ☐ ……

115.Casts/Crystal - ☐ ☐ ……

MOTION: Present (1) Absent (2)

116.Ova - ☐ ☐

117.Cyst - ☐ ☐

118.Occult blood - ☐ ☐

119.Pus cells - ☐ ☐

INCLUSION CRITERIA: Yes(1) No(2)

120.Vitiligo patient. ☐ ☐

121.Age between 5 to 12 years. ☐ ☐

122.Willing to be admitted as In patient in our ward for 48 days or willing to attend OPD once in 7 days for 48 days. ☐ ☐

123.Willing to give blood specimen and photograph before and after treatment. ☐ ☐

EXCLUTION CRITERIA: Yes(1) No(2)

124.Jaundice ☐ ☐

125.Hypopigmended patches of leprosy and burns ☐ ☐

126.Connective tissues disorders

127 Cerebral palsy

128.Cardiac disease

129.Admitted to trail: Yes(1) ☐ No(2) ☐

130.If yes, a) S.No: ☐☐ 149.IP:☐ OP: ☐

131.Drugs issued for O.P.Patients:

1.No.of packs:_____

2.:Weight of pattru _____

132..Date:……

133.Station:……. Signature of Doctor

A PILOT OPEN CLINICAL TRIAL OF LAGU SEENA CHOORANAM AND VENPANDAI PATTRU FOR THE TREATMENT OF *VENPADAI (*VITILIGO)

FORM II-ASSESSMENT PROFORMA

1. OP/IP No:_____ 2.BED No:_____3.S.No:_____

4. NAME:_____

5. DATE OF ADMISSION: ☐☐☐☐☐☐

6. DATE OF ASSESSMENT: ☐☐☐☐☐☐

7. DAY OF ASSESSMENT: ☐☐

CLINICAL ASSESSMENT CHART:

8. ANATOMICAL LOCATION…….

9. COLOUR Normal ☐ Hyperpigmented ☐ Hypopigmented ☐

10. SIZE OF THE LESION (Length cm):……

11. SHAPE: Irregular ☐ Round ☐ Dispersed ☐

12. PRURITUS: Present ☐ Absent ☐

13. SWELLING: Present ☐ Absent ☐

14. ERYTHEMA: Present ☐ Absent ☐

15. DEPIGMENTATIOPN OF HAIR

Present ☐ Absent ☐

16. SENSATION

Normal ☐ Paraesthesia ☐ Numbness ☐

Painful ☐ Burning ☐ Pricking ☐

17. SCALING: Present ☐ Absent ☐

18. CRUSTING: Present ☐ Absent ☐

19. OOZING: Present ☐ Absent ☐

20. MACULES: Present ☐ Absent ☐

21. PAPULES: Present ☐ Absent ☐

22. VESICLES: Present ☐ Absent ☐

23. PUSTULES: Present ☐ Absent ☐

24. PALPATION:

Normal ☐ Smooth ☐ Rough ☐

Warm ☐ Cold ☐

25.PIGMENTATION : Present ☐ Absent ☐

……..

26.Naadi…….

LAB INVESTIGATION :(Only on Day 21and Day 48)

BLOOD:

27. TC (Cells/Cu mm) ☐☐☐☐

DC (%) 28.N- ☐☐ 29.L- ☐☐ 30.M- ☐☐ 31.E - ☐☐ 32.B- ☐☐

ESR (mm) 33.1/2 hr- ☐☐ 34.1 hr- ☐☐

35. Hb (gm %)- ☐☐☐☐

URINE: Present (1) Absent (2)

41.Albumin ☐ ☐…….....

42.Sugar ☐ ☐……

Deposit Present (1) Absent (2)

43.Pus cells - ☐ ☐ ……

44.Epithelial cells - ☐ ☐ ……

45.Red cells - ☐ ☐ ……

☐ ☐

MOTION: Present (1) Absent (2)

47. Ova - ☐ ☐

48. Cyst - ☐ ☐

49. Occult blood- ☐ ☐

50. Pus cells - ☐ ☐

51. NEERKURI……

52. NEIKURI: Vatha neer(1)☐ Pitha neer(2) ☐ Kaba neer(3)☐

53. RESULT: Cured ☐ Improved ☐ No change ☐

FOR O.P.PATIENTS:

54. Drugs issued:

1. No. of pack……

2. Weight of pattru _____

55. Date:

56. Station: Signature of Doctor

NATIONAL INSTITUTE OF SIDDHA, CHENNAI-47
AYODHIDOSS PANDITHAR HOSPITAL
DEPARTMENT OF KUZHANTHAI MARUTHUVAM
A OPEN CLINICAL TRIAL OF SIDDHA DRUGS *LAGA SEENA CHOORANAM* AND *VENPADAI PATTRU* IN THE TREATMENT OF *VENPADI(*VITILIGO)
CONSENT FORM

Certificate by Investigator

I certify that I have disclosed all details about the study in the terms readily understood by the patient.

Date: _____ Signature: _____

Name: _____

Consent of Informant

I have been informed to my satisfaction, by the attending physician, the purpose of the clinical trial, and the nature of drug treatment and follow-up including the laboratory investigations to be performed to monitor and safeguard my Son/Daughter body functions.

I am aware of my right to opt out of the trial to my Son/Daughter at any time during the course of the trial without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included my Son/Daughter as a subject in the clinical trial of *Lagu seena Chooranam* and Venpadai pattru in the treatment of *Venpadai*(Vitiligo).

Date: Signature

Name:

Relationship:

Date: Signature of witness:

Name:
Relationship:

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IN PATIENT DEPARTMENT

S NO.	Name	Age/Sex	IP NO	DOA	DOD	RESULT
1.	K. Subbulakshmi	9/f	241	24.9.08	29.10.08	Moderate
2.	K. Manjubharathi	12/f	243	24.9.08	26.10.08	Good
3.	Akash	9/m	247	26.9.08	15.10.08	Moderate
4.	Siva	6/m	249	27.9.08	1.11.08	Moderate
5.	Prabhu	12/m	253	29.9.08	16.10.08	Good
6.	Kavitha	10/f	245	26.9.08	17.10.08	Moderate
7.	Jagan	6/m	246	26.9.08	17.10.08	Mild
8.	Hari	8/m	255	29.9.08	9.10.08	Moderate
9.	Latha	11/f	262	4.10.08	18.10.08	Good
10.	Inbarasi	6/f	266	7.10.08	29.10.08	Moderate
11.	Yaseen Beevi	8/f	269	8.10.08	29.10.08	Moderate
12.	Akshaya	6/f	272	10.10.08	28.10.08	Good
13.	Syedh	9/m	274	14.10.08	30.11.08	Good
14.	Sobana	11/f	276	20.10.08	31.11.08	Moderate
15.	Priya	12/f	282	29.10.08	9.11.08	Good
16.	Kavya	10/f	285	31.10.08	6.11.08	Moderate
17.	Prasanth	8/m	295	5.11.08	17.11.08	Good
18.	Sivanathan	6/m	326	21.11.08	18.12.08	Moderate
19.	Tejasri	7/f	330	22.11.08	9.12.08	Good
20.	Ameer	8/m	332	24.11.08	8.12.08	Good

The patient who are treated in IP Department after having slight improvement and discharged, then continued to OP Department.

OUT PATIENT DEPARTMENT

S NO	NAME	Age/ Sex	IP NO	DOA	DOD	Result
1.	Sivaranjani	8/f	AM1932	20.8.08	8.10.08	Moderate
2.	Sarasvathi	12f/	AM1978	20.8.08	8.10.08	Moderate
3.	Sowendhariyan	12/m	AM1872	20.8.08	8.10.08	Good
4.	Nithyasri	10/f	AM2847	23.8.08	11.10.08	Mild
5.	Saber Satik	9/m	AM2528	23.8.08	11.10.08	Good
6.	Yaseen Beevi	8/f	AM2603	23.8.08	11.10.08	Moderate
7.	Deepak	10f/	AM2523	23.8.08	11.10.08	Good
8.	Varshini	7/f	AM2579	23.8.08	11.10.08	Good
9.	Rajkumar	12m/	AM2521	23.8.08	11.10.08	Good
10.	Pandian	12/m	AM2634	23.8.08	11.10.08	Good
11.	Akshaya	10/f	AM2606	23.8.08	11.10.08	Good
12.	Sangeetha	6/f	AM3302	25.8.08	13.10.08	Moderate
13.	Janani	12/f	AM4669	30.8.08	18.10.08	Good
14.	Ameerbasha	8/m	AM4615	30.8.08	18.10.08	Moderate
15.	Rohith	8/m	AM4859	31.8.08	19.10.08	Moderate
16.	Divya	6/f	AM4872	31.8.08	19.10.08	Good
17.	Sudha	11/f	AM5886	31.8.08	19.10.08	Good
18.	Santhakumar	11/m	AM4822	31.8.08	19.10.08	Moderate
19.	Vaishnavi	10/f	AM8508	15.9.08	28.10.08	Good
20.	Kirankumar	9/m	AM9682	19.9.08	2.11.08	Good
21.	Kalaimathi	11/f	AN70	20.09.08	8.11.08	Good
22.	Inbarasi	7/f	AN47	20.09.08	8.11.08	Moderate
23.	Shivananthan	6/m	AM9954	20.09.08	8.11.08	Good
24.	Arunsai	8/m	AN811	23.9.08	11.11.08	Good
25.	Parkavi	9/f	AN1025	23.9.08	11.11.08	Good
26.	Harini	8/f	AN1183	24.9.08	12.11.08	Moderate
27.	Dinesh	12/m	AN3767	27.9.08	21.11.08	Mild
28.	Krithika	7/f	AN1278	29.9.08	15.11.08	Good
29.	Harinath	8/m	AN2416	2.10.08	17.11.08	Good
30.	Thilaga	11/f	AN3516	3.10.08	20.11.08	Moderate